STUDIES ON RESISTANCE TO MILDEW *(Erysiphe graminis)* AND LEAF BLOTCH *(Rhynchosporium secalis)* IN BARLEY

by

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DECLARATION

This thesis has been composed by myself and describes experimental work which I carried out between October 1976 and August 1980.

CONTENTS

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Abstract.

A major consideration in the use of genetical resistance against infection of crop plants is the ability of the pathogen concerned to evolve genotypes with virulence to overcome such resistance. This applies particularly to oligogenic forms of resistance with major effects and it is possible that forms of partial resistance may be durable and have economic benefit. This study was concerned with the development of screening methods and identifying possible sources of partial resistance in barley to two fungal foliar pathogens, *Erysiphe graminis* f.sp. *hordei*, causal agent of mildew and *Rhynchosporium secalis* causing leaf blotch.

With respect to barley mildew, lines from Ethiopia, Turkey and Israel, as well as lines and cultivars from two European Barley Disease Nurseries, were found in preliminary work to exhibit a wide range of response when exposed to natural inocula of *E. graminis*. A screening procedure was adopted to favour the selection of virulences, from the pathogen population, for particular host genotypes and indicated those lines which gave consistently low disease levels. When these lines were tested in conjunction with commercial cultivars against known isolates of *E. graminis* with various virulence combinations, different patterns of resistance response were evidenced. Firstly, vertical resistance was associated with commercial cultivars but not lines; secondly, consistently high resistance was shown by some lines indicating resistance factors other than those apparently present in most existing commercial cultivars and thirdly, some lines consistently showed intermediate levels of horizontal resistance. In tests on commercial cultivars grouped according to their barley mildew resistance categories, both intra- and inter-group differences were recorded. Variations in group characteristics between years was

attributable to changing virulence combinations in the pathogen population. Variations within groups were low and inconsistent between assessments: in some cases adult plant resistance may have been important. The reported tolerance of Proctor may be associated with delayed infection of emerging leaves and little necrosis resulting from infection. Microscopic assessments indicated that leaf position and plant age may influence fungal development, although there were no apparent qualitative differences in pathogen behaviour on cultivars evidencing varying degrees of partial resistance.

Studies on *R. secalis* were hampered by difficulties in ensuring epidemic development in screening tests, although the development of a system based on automatic misting equipment eventually overcame these and susceptible cultivars became rapidly infected. In glasshouse trials on a range of lines and cultivars, infection above a threshold level did not lead to increased impairment of leaf functioning: growth habit may be of some importance in determining cultivar susceptibility to this disease. It was demonstrated that lesion development and level of spore production for different cultivars may not be correlated, the level of spore production being epidemiologically significant in the field.

INTRODUCTION

AND

LITERATURE REVIEW

INTRODUCTION

In a "natural" plant community, host and pathogen exist in an apparent equilibrium where the diversity of vegetation prevents a rapid build up of extensive disease epidemics. Efficient crop production systems, however, involve the cultivation of dense, uniform plant populations. In the case of cereals, one cultivar may be grown exclusively and intensively in large areas. Thus, an almost constant source of host material is available for colonisation by the pathogen which can proliferate and cause serious crop yield losses.

Present day fungicides can effectively control many air-borne diseases in cereals (A.D.A.S., 1981). However, the use of chemicals increases the cost of production and any increase in yield as a result of reduced infection must be balanced against the cost of application. Moreover, widespread use of chemicals is an undesirable source of environmental pollution (Russell, 1978). Many recently introduced chemicals may become less effective when widely used and selection pressures result in the emergence of fungicide insensitive strains (Wolfe, 1972).

Genetical control of disease, through the use of resistant cultivars, presents no hazard of pollution but cultivars must be bred with regard to other factors, notably yield and quality in addition to resistance against a broad range of pathogens. A major problem in resistance breeding is genetical variation in the pathogen populations and strategies to produce durable forms of resistance, effective against the total genetical range of a pathogenic species, now receive considerable attention.

Resistance may be based on one or a few genes (oligogenic) or on many genes (polygenic). Oligogenic resistance may prove very

effective in a mixed cropping system or when a disease increases in a "simple interest" analogy (Van der Plank, 1963). However, where monoculture prevails with a rapidly reproducing pathogen, selection of a population with the virulence genotype to overcome resistance in the host is readily encouraged (Flor, 1955). A cultivar with a high level of polygenic resistance may have a constant low level of disease, which may be acceptable in a crop if it does not significantly affect the yield. Also, as many genes are involved it is unlikely that resistance will be rapidly overcome (Wolfe, 1972). The levels of disease in a crop with this sort of resistance may, however, increase as the pathogen population gradually adapts to the resistance present (Wolfe and Schwarzbach, 1978).

The extent of the problem presented by genetical variation in the pathogen is influenced to a large degree by the biological characteristics of the organism. Those pathogens which produce several generations of air-borne spores in large numbers during a single growing season will have the capacity to show rapid genetical changes in populations as they respond to selection. This type of pathogen will overcome oligogenic resistance very rapidly whereas a pathogen which produces few spores and has a more restricted method of dispersal may be effectively controlled by single gene resistance.

A further consideration relating to resistance is the nature of the mechanisms of both infection and defence involved, based on the type of association between the host and pathogen. In a highly specialised relationship where the pathogen is a strict biotroph (Luttrell, 1974), rapid death of host tissue, as in the hypersensitive reaction, may serve to isolate the pathogen before it can sporulate and perpetuate itself. Here successful infection results from the

establishment of an apparently specific nutritional relationship between the metabolic systems of the host and pathogen: factors leading to early host cell injury appear to impair this relationship. Necrotic responses are thus linked to resistance mechanisms in some measure and appear to be particularly involved with race-specific resistance.

A hemibiotroph or necrotroph presents a host relationship which differs in certain basic ways from those of a biotroph (Luttrell, 1974). The pathogen may derive nutrients from dead or dying host cells and substantial enzyme or toxin activity play an important part in disease development. Resistance to diseases caused by such pathogens may depend, in part, on resistance to this biochemical degradation.

In considering resistance mechanisms, information on patterns of disease development on plants throughout their growth cycle and on the development of the pathogen within host material is needed (Zadoks, 1971). In addition, a knowledge of the economic loss caused by a disease at any given level of infection is needed for the practical application of disease resistance in farming today (Russell, 1978).

In the present study, various aspects of disease resistance in relation to foliar pathogens of barley were considered, aiming to provide a further understanding of host parasite relationships as a basis for a more effective use of genetical resistance. The investigations involved mainly barley mildew (*Erysiphe graminis* D.C. ex Merat f.sp. *hordei* Marchal) and were carried out with existing and past commercial cultivars as well as with lines from various barley collections of different sources. Some investigations were also carried out on barley leaf scald or blotch (*Rhynchosporium secalis* (Oud.) J.J. Davis) in studies additional to the main work on *E. graminis*.

LITERATURE REVIEW

The Disease and the Pathogens.

Powdery mildew (*Erysiphe graminis f.sp. hordei*) is the most widespread and serious disease of barley today. In Britain it may cause losses of up to 35% (Rea and Scott, 1973) in years of severe disease levels, although a 10% yield reduction may be more common (Brooks, 1972). The causal fungus, *Erysiphe graminis*, develops as a superficial mycelium over the aerial surface of the plant and haustoria penetrate the epidermal cells. Photosynthetic activity is impaired, while respiratory and transpiration loss is increased as a result of infection of the shoot tissues (Habeshaw and Lennard, 1978). Field losses due to mildew are brought about in two main ways: an early attack may restrict root growth, cause a reduction in the number of fertile tillers and adversely affect the overall size of the plant, its leaves and its ears (Last, 1962); attacks later in the growing season may result in poor filling of ears and shrivelled grain.

In comparison with mildew, leaf blotch or scald (*Rhynchosporium secalis*) is not a frequent or widespread problem in Britain: national yield losses may average 1% to 2% annually (James, 1969). Local outbreaks, however, can be very damaging and on occasion may cause yield reductions equal to those resulting from a mildew attack (Jenkins and Jemmett, 1967). Leaf scald mainly affects the leaf blades but may also occur on leaf sheaths and on the ear itself. The pathogen mainly colonises the subcuticular region of the leaf but in later stages of infection may penetrate further into the tissues. Hyphae rarely penetrate into the cells, but absorb nutrients through the cell

walls which eventually collapse. As with mildew infected material, there is a reduced photosynthetic activity and increased respiration and transpiration loss. In Britain, attack by leaf scald early in the year is not usually severe. At later growth stages, however, an attack may result in considerable damage to the upper leaves particularly on the flag leaf, causing yield loss through poor grain setting and filling (Doling, 1964; James, Jenkins and Jemmett, 1968).

The mildew fungus, *E. graminis*, has the capacity to produce several generations of conidia during a single growing season. The air-borne spores are rapidly dispersed both through and between crops causing serious epidemics (Cherewick, 1944). Ascospores, the result of the sexual process, are formed towards the end of the growing season being dispersed in late summer and early autumn. The production of fertile ascocarps is favoured by high humidity. Conidial production is favoured by dry conditions and free water may inhibit the germination of newly deposited spores (Manners and Hossain, 1963). The fungus generally overwinters in a quiescent mycelial state on living tissues of susceptible plants. The life cycle is summarized in Figues 1 and 2.

In the case of *R. secalis*, 100% relative humidity or free water is required for the successful germination of spores (Ayres and Owen, 1970). Dispersal of *R. secalis* is water aided; Priestly (1972) described the dislodging of spores within a lesion by rain drops and subsequent spread within the crop by splash droplets. Skoropad (1960) referred to the spread of disease between crops by wind-borne rain-splash but Ayesu-Offei and Carter (1971) were unable to confirm this. *R. secalis* may over winter on stubble and other plant debris as well as on living plants; cultural practices may account for some spread of disease between crops (Stedman, 1977). There have been reports

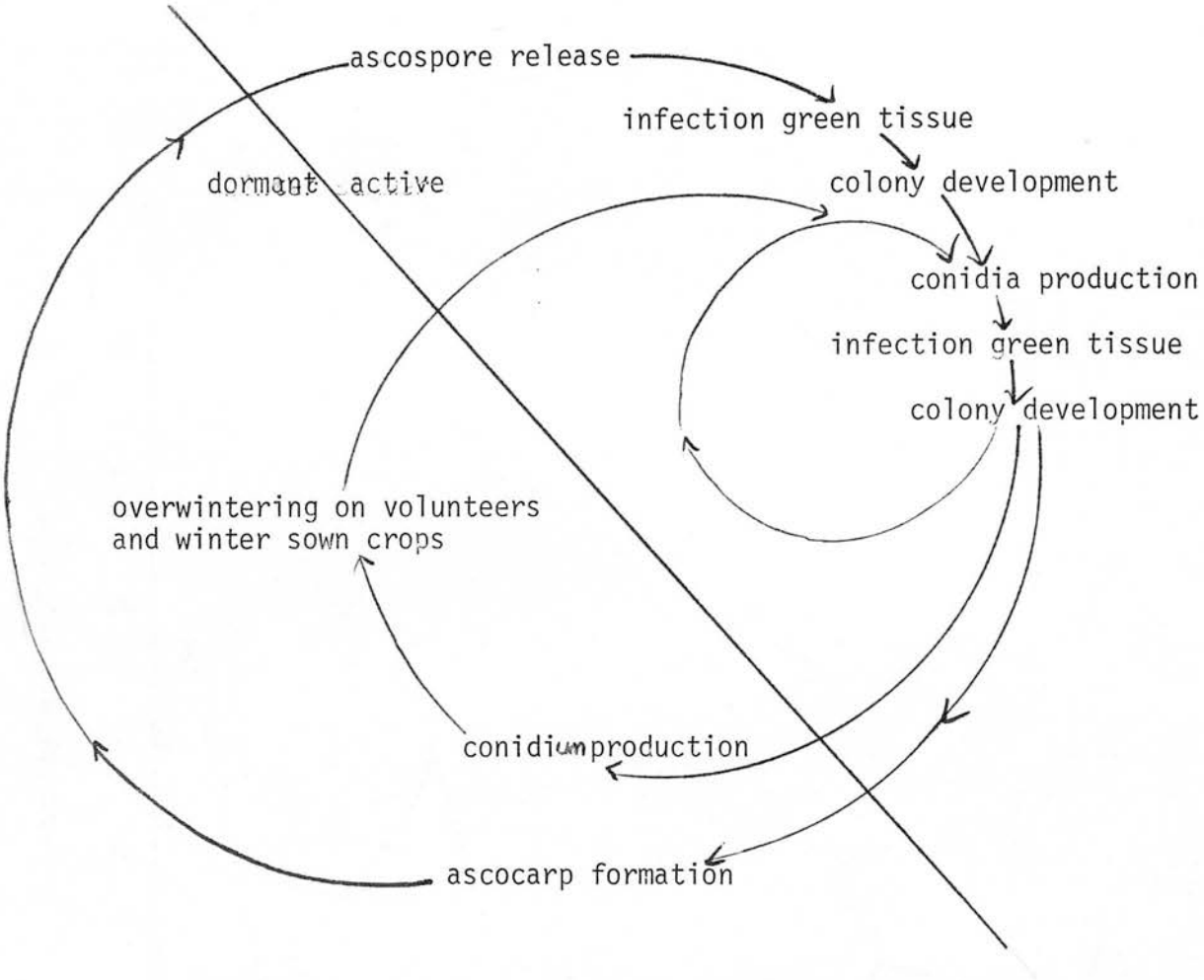


Figure 1. Life cycle of Erysiphe graminis.

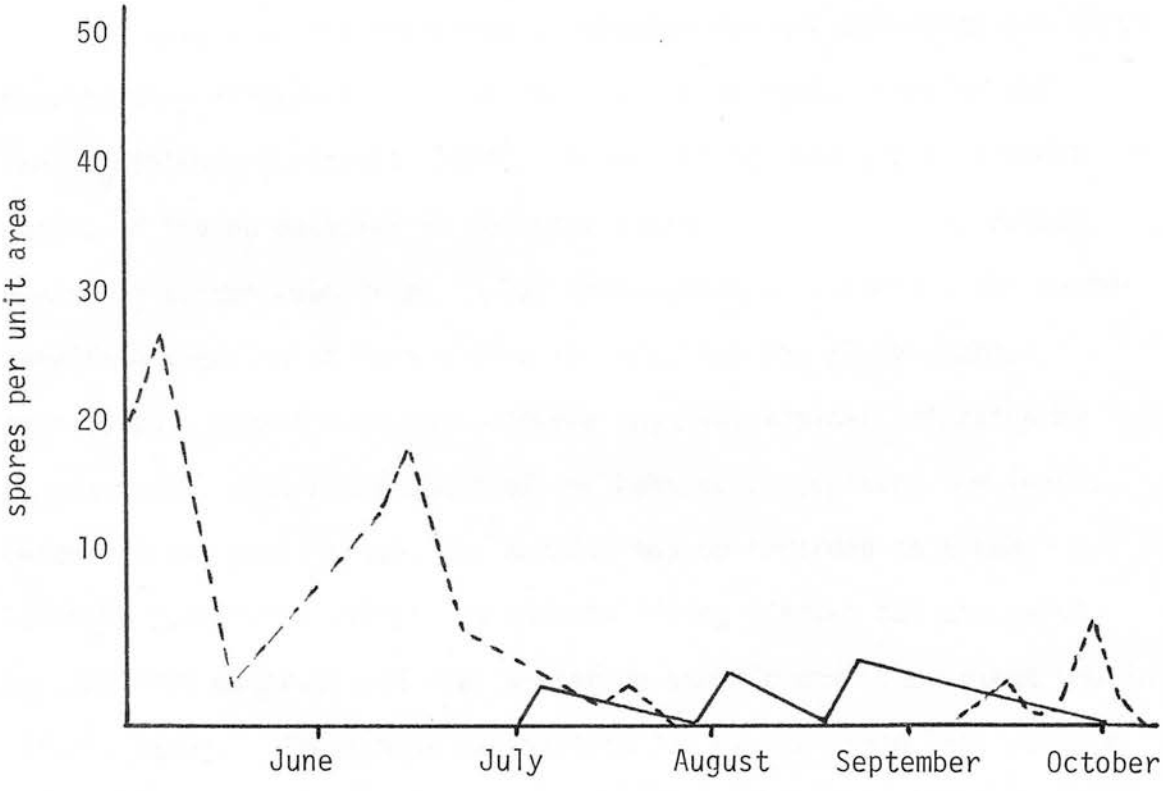


Figure 2. Seasonal production of conidia and ascospores. (based on Turner, 1956).

--- conidia (1950)

——— ascospores (1950)

of *R. secalis* transmission on infected seed (Habgood, 1971; Kay, 1971; Kay and Owen, 1973; Jackson and Webster, 1976) which may be important in carry-over and infection between crops. The uncertain methods of dispersal and exacting environmental requirements probably account for the sporadic occurrence of leaf blotch in Britain except in the south-west of England, where favourably moist and warm conditions promote infection in most years. The life cycle is summarized in Figure 3.

The nature of the relationship between the two pathogens and their host is very different. *E. graminis* is an obligate parasite and strict biotroph (Luttrell, 1974): it therefore requires a constant supply of living material to colonize and over winters on volunteer plants or autumn-sown crops. For this reason of biotrophy the hypersensitive reaction of host plants to infection has always been considered a significant host defence reaction against infection by *E. graminis*. The rapid death of the host cells isolates the fungus before it can proliferate. *R. secalis* may be regarded as a hemibiotroph (Luttrell, 1974): it infects living tissues but can exist on dead host material and over winter on stubble and other plant debris (Evans, 1969). The fungus may destroy 75% of the green leaf surface and still sporulate efficiently (Ayesu-Offei and Carter, 1971). In the case of *R. secalis* enzymes play an important part in disease development and toxin production may be involved in leaf death (Ayesu-Offei and Clare, 1971; Jones and Ayres, 1972). The toxin, rhynchosporoside, appears to be host selective, susceptible barley lines being sensitive to the chemical. Not all lines resistant to *R. secalis* infection are, however, insensitive to rhynchosporoside (Auriol, Strobel, Beltran and Gray, 1978). The influence of *E. graminis* on leaf senescence in susceptible lines is, in contrast, uncertain (Finney, 1979) and possibly relates to stress factors.

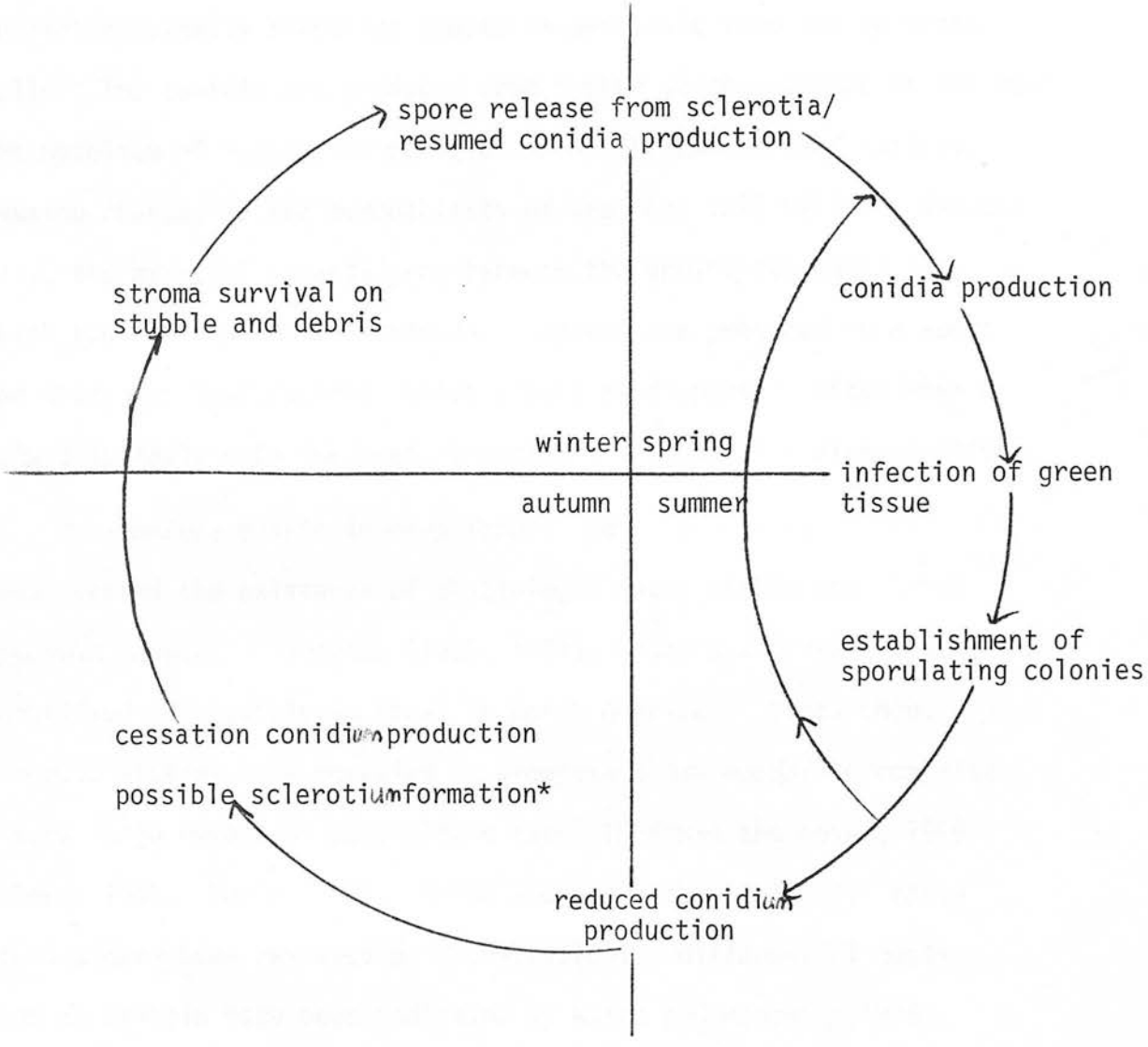


Figure 3. Life cycle of Rhynchosporium secalis.

* Shipton, (1974).

The two fungi effect colonization of the host in different manners. *E. graminis* forms a surface mycelium and only the characteristically branching haustoria penetrate into the epidermal cells. The conidia are produced from hyphae on the surface of the leaf. The mycelium of *E.graminis* ramifies mainly below the leaf cuticle, causing changes in the permeability of the host cell walls. Eventually, the mycelial strands grow between the underlying cells into which knob-like branches protrude. Spores are produced in a spore bed under the leaf cuticle, which breaks at dispersal, often when a rain drop falls onto the leaf, exposing the spores for dissemination.

E. graminis exists in many forms; Mains and Dietz (1930) demonstrated the existence of physiologic races within the forma specialis *hordei*. Moseman (1955, 1959), using six differentials, identified 22 physiologic races in North America. Since then, numerous studies have revealed *E. graminis* f.sp. *hordei* to comprise a very large number of physiologic races (Hoffman and Nover, 1959; Wiberg, 1962; Wolfe, 1967; Wolfe and Schwarzbach, 1975): these studies have been reviewed by Hiura (1978). Differential hosts used in Britain have been indicated by Wolfe and Minchin (1976). A systematic survey of physiologic races throughout the British Isles was begun in 1967 and has continued annually. (Anon. 1968; Wolfe, 1969; 1970; 1971; 1972; 1973; Wolfe and Wright, 1974; 1975; 1976; 1977; 1978; Wolfe and Slater, 1979; 1980; Wolfe, Slater and Minchin, 1981). The reports indicate the emergence of newly recognised virulences, changing host resistance and the expansion of host differential series in recent years.

Physiologic specialization is less well documented in the case of *R. secalis*. Owen (1963) demonstrated variability in the pathogen

but this was thought to be due to a lack of uniformity in experimental conditions. Owen (1968) identified two races from a survey carried out in 1967 in Britain; these were designated U.K.1 and U.K.2 (Owen, 1969). Fowler and Owen (1971) also reported two distinct races found in British barley cultivars: one isolate attacked all cultivars with which it was challenged and one isolate appeared to be less virulent on Dea and Pioneer than on other cultivars. Williams and Owen (1973) using 12 cultivars of barley and 122 single spore isolates of *R. secalis* again reported on the two races designated U.K.1 and U.K.2. Differentiating cultivars were reduced from 12 to 3, Cambrinus (later amended to Deba Abed) Dea and Osiris. Clifford and Jones (1978) used five differential cultivars, Maris Mink, Triumph, Armelle, La Mesita and Magnum; more recently in 1979, identifying four races (Jones and Clifford, 1980). In 1980 the number of designated races increased to five (Jones and Clifford, 1981). Schein (1960) differentiated seven pathogenic races in America using a set of six differential cultivars. Ali, Mayfield and Clare (1976) identified 35 pathotypes in Australia: only one barley cultivar, Atlas 46, was resistant to them all.

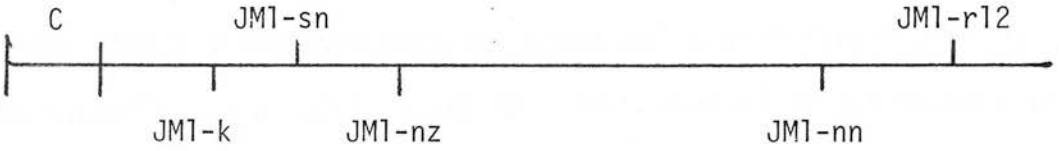
The Host.

In 1955, Mŏseman screened the then complete world collection of barley, comprising 6,273 entries, for resistance to mildew. He located 128 lines with resistance to eight physiologic types of *E. graminis* collected in North America. These lines showed an almost immune response suggesting a race specific resistance. Since then, various collections of barley have been screened (Ralski and Mikolajewicz, 1969; Fischbeck and Schwarzbach, 1972; Brückner, 1975).

Caddel (1976) tested 10,420 lines from the U.S.D.A. Small Grains Collection for resistance to a natural mildew population in Morocco. Entries from Turkey were found to occupy the middle position in being moderately resistant to moderately susceptible. These responses tend to indicate a low level of polygenic resistance. Caddel found lines from Ethiopia generally resistant when challenged with this population; when later tested in America the same lines were not resistant to American pathogenic populations, illustrating the dependency of resistance expression on the virulences present in the natural population.

Biffen (1907) first studied the inheritance of resistance in barley to infection by *E. graminis:* he showed that a single recessive gene conferred resistance in the wild barley *Hordeum spontaneum*. Since then numerous studies have been carried out and extensively reviewed (Schaller and Briggs, 1955; Hiura, 1960; Moseman, 1966; Wolfe, 1972b; Wiberg, 1974a, 1974b). There are now thought to be at least seven loci conditioning resistance to *E. graminis* although not all of these have been used in commercial breeding programmes. (Figure 4). At the Ml-a locus, 11 alleles conferring resistance have been identified designated Mla_1 to Mla_{11} (Moseman and Jørgensen, 1972; Jørgensen and Moseman, 1971). Those genes reported to be of present day economic importance are Mla_4 (from HOR 1063), Mla_7 (from Lyallpur 3465); Mla_9 (from Monte Cristo); also Mlg from Goldfoil, Mln from Hanna and Mlp from Nigrate. In addition, two resistance genes, Mlh and Mlk, derived from Ragusa lines have been used in winter barleys and incomplete forms of resistance from *Hordeum laevigatum* have been exploited in cultivars including Vada, Minerva and Universe (Wolfe, 1972b, Russell, 1978). An induced mutant which has now been found

Figure 4. Reistance loci on long arm of chromosome 4. (Wolfe, 1972).



JM1-sn = M1a (11 alleles)

M1-a	Algerian
M1-a2	Black Russian
M1-a3	Ricardo
M1-a4	No. 22
M1-a5	Gopal
M1-a6	Franger
M1-a7	Line 4831
M1-a8	Heils Hanna 3
M1-a9	Monte Cristo
M1-a10	Durani
M1-a11	A222

to occur naturally in some Ethiopian lines, has several resistance alleles at the mlo locus on chromosome 4 (Jørgensen, 1971; 1976a; 1976b). Lines with this mutation show a chlorotic and necrotic spotting on the leaves that is usually associated with low yields (Russell, 1978). Various workers (Benada and Brückner, 1964; Benada, Dušek and Novak, 1967) suggested that this necrotic flecking was associated with the hypersensitive reaction response. Later, Benada (1969) determined that the brown cells were discoloured by the accumulation of phenols but the cells were alive and capable of supporting mildew development. In some lines 95% of the leaf area was affected and a reduction in the photosynthetic efficiency of the leaf ensued. This type of resistance has not, therefore, been exploited in a breeding programme as yet. Other resistances associated with commercial lines have been assigned to groups with resistance factors R0 to R8 singly or in combination, depending on the resistance genotype (Wolfe, Slater and Minchin, 1981). Group R0 contains those cultivars with no known major genes for resistance; Group R1 contains mainly winter cultivars and Group R2 - R8 contain mainly spring cultivars which have been successively introduced as groups have become susceptible to *E. graminis* attack.

The pathogen population in Britain as a whole has been directly affected by the introduction of race specific resistances (Wolfe and Barret, 1976), as indicated in the following summary based on accounts by Wolfe and Schwarzbach (1978):-

Resistance Group	
R0	Cultivars in this group have no known major genes for resistance and are no longer in common usage except for Golden Promise and Proctor. Pathogen populations have been generally little affected by the introduction of these cultivars although there may be evidence for Proctor to have precipitated a move away from V5 virulences
R1	The resistances in this group are mainly conditioned by Mlh genes (Ragusa b) and were widely introduced into winter barley cultivars after World War II. V1 virulences are now common in both winter and spring populations, perhaps reflecting the importance of winter cultivars in the over wintering of *E. graminis*.
R2	Cultivars with Mlg (Pflugs Intensiv) resistance did not increase greatly in popularity until after World War II, although Pflugs Intensiv was first recognised officially as an original cultivar as early as 1921. The widespread use of cultivars with this resistance led to a great increase in pathogen populations with the corresponding V2 virulences. For example cultivars such as Zephyr and Julia were grown extensively in the late 1960's and early 1970's respectively and pathogen populations with V2 are now common.

Resistance Group	
R3	Resistance conditioned by Mla6 factors from a line of *Hordeum spontaneum nigrum* provided a second widely used source of resistance in spring barley. Introductions of cultivars such as Maris Badger (Mla6) and later Impala (Mlg + Mla6) led to an increase in combined virulences in the pathogen population.
R4	Resistance Mlv derived from *Hordeum laevigatum* has not been used extensively but cultivars such as Vada and Mala Abed have been grown in small areas for a long time. As a result, although the V4 factor is common in many pathogen populations it is at too low a level to produce large mildew epidemics.
R5	Cultivars with the "Arabische" source of resistance (Mlas) became generally available in the late 1960's. The introduction of cultivars such as Sultan and Hassan led to the emergence of V5 and also V(2+5) virulences as R2 lines were still commonly grown and V2 virulences very plentiful. There was a higher frequency of V(2+5) virulences on R5 plants than on R2 as the acreage of the former was insufficient to boost V5 virulences on R2 plants.

Resistance Group	
R6	Cultivars with resistance genes M1a4/7, "Lyallpur" resistance, were developed commercially from the 1960's often in association with M1g (e.g. Mazurka). Selection for virulence has parallelled that for M1as. Populations with combined virulences to M1as and M1a4/7 are, however, still rare.
R7 R8	Cultivars in these groups, e.g. Tyra, M1a (R7) and Simon, M1a4/9 (R8) have been too recently introduced to have greatly affected the pathogen population. R6 plants already show greater infection than when they were first introduced, reflecting an increased frequency of V6 virulences. As more cultivars in resistance groups R7 and R8 are grown over a greater area it is likely that V7 and V8 virulences will also increase.

In considering further cultivars introduced with a combination of resistances, varying results have appeared. When Impala with a combination of R2 and R3 resistances was introduced, cultivars with resistances R1 or R2 or R3 only were commonly grown. Selection of a pathogen with V(2+3) was, therefore, rapid and Impala, initially highly resistant, quickly became completely susceptible. More recent introductions with combined resistances, e.g. Maris Mink, R(2+5+x), Aramir R(2+5), Abacus R(2+4), and Mazurka R(2+6) have remained largely resistant: as a range of resistance groups are now in wide use and the application of chemical sprays is common, the build-up of one particular virulence to epidemic proportions is less favoured.

The mechanisms by which certain genes exert their effect has been studied by Ellingboe (1972) working with four mildew resistance genes, Mla, Mlg, Mlk and Mlp. These were challenged by four races of *E. graminis* possessing corresponding virulences. Studies following the development of single spore colonies showed that even in incompatible host-parasite combinations a small proportion of fungal spores developed into sporulating colonies, although the frequency and vigour of the colonies varied a great deal. There appeared to be two stages in the resistance process (Figure 5):

i) A high percentage of spores, although germinating and forming germ tubes, normally failed to establish haustoria, thus preventing the proliferation of a surface mycelium.

ii) After the initial check, development was hindered at a second stage of development according to the resistance gene present.

Usually the host cells collapsed in a hypersensitive reaction associated with major gene resistance. A study of 11 mutant alleles at the ml0 locus (Jørgensen and Mortensen, 1977) indicated that haustorial development was impaired as a first stage of host resistance.

The *E. graminis* resistance discussed so far has been the response to major gene, race specific factors. However, within each group, cultivars which have resistance at the same locus may show considerable variation in *E. graminis* infection. For instance, within group R2, Deba Abed, Swallow and Union were shown to be significantly more resistant to isolates of *E. graminis* with corresponding virulences than were Zephyr or Mosane (Howard, Johnson, Russell and Wolfe, 1970). Variation in resistance has also been shown amongst cultivars in group R0, those with no known major genes for resistance (Jenkins and Storey, 1975). There is thus evidence for a background resistance which may

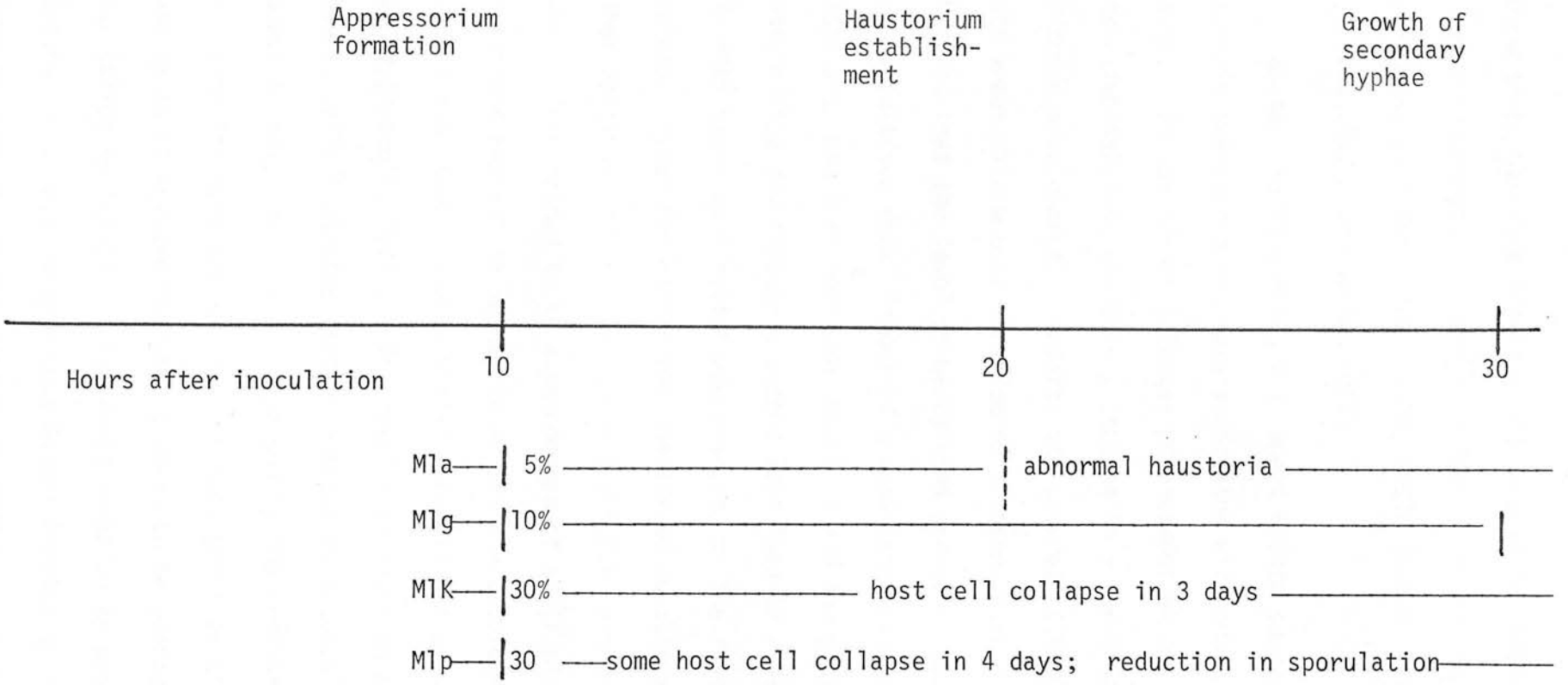


Figure 5. Time after inoculation by race with corresponding avirulence gene when 4 major resistance genes in barley: Mla, Mlg, Mlk, Mlp, inhibit powdery mildew infection. (based on Ellingboe,1972).

prove to be non-race specific and durable in comparison to those already discussed. These incomplete forms of resistance are termed horizontal (Van der Plank, 1963), field (Black, 1960) or non-race specific (Hayes and Jones, 1966).

Shaner (1973) working with wheat mildew suggested that non-race specific resistance was more associated with adult plants than seedlings. It may affect pathogen development at any stage of infection and establishment, ultimately leading to a reduction and/or delay in epidemic development. Roberts and Caldwell (1970), also working with wheat mildew over a period of fifteen years from 1954 to 1969, observed that the level of background non-race specific resistance in the cultivar Knox "remained undiminished", although major gene resistance had been overcome both in field and glasshouse conditions. Pustule size and frequency seemed less than in other cultivars with the same major gene resistance overcome by the relevant virulent isolate. Thus resistance was considered durable and transferred to other cultivars during the Purdue - U.S.D.A. breeding programme at that time. This seemed to be a phenomenon of adult plants. In oats, background resistance seemed to affect the development of haustoria (Carver and Carr, 1975), whilst generation time was retarded in barley (Russell, 1976). The results of tests on models (Parlevliet and Zadoks, 1977) indicated that horizontal resistance controlled by a number of polygenes may be expressed by the individual genes acting on a gene-for-gene basis with virulence genes of the pathogen. In some cases background resistance seems to be expressed as less yield loss following infection than would normally be predicted. Proctor appears to be less damaged than Golden Promise by *E. graminis*, although both are in resistance group R0, and is said to show "tolerance"

to the disease (Little and Doodson, 1972). Asse, also in resistance group RO, has also exhibited considerable tolerance to *E. graminis* in the field (Frimmel, Schwarzbach and Fischbeck, 1975). Russell (1978) defines a tolerant plant as one which "is attacked to the same degree as other plants, but which suffers less damage (in terms of yield or quality) as a result of the attack".

There have been fewer screening programmes to assess resistance of cultivars to *R. secalis*, than there have been to assess *E. graminis* resistance. Mackie (1929) first studied the inheritance of resistance to *R. secalis* using an unnamed cultivar. He concluded that resistance was controlled by a single recessive gene. It has since been suggested that a multi-allelic series of genes may be involved in resistance to *R. secalis* (Hapgood and Hayes, 1971; Ali, 1975a; 1975b), the expression of resistance being dependent on environmental conditions, with the exception of Armelle which is always resistant (Hapgood, 1977). Williams and Owen (1973) suggested that conflicting results from many studies on resistance to *R. secalis* may be due to non-uniform environmental condition. Shipton (1974) reviewed the genetics of resistance to *R. secalis* and summarized the position regarding the designation of resistance genes.

Identifying resistance.

The development of disease on a plant is influenced by many factors. The environment may influence the activity of the pathogen and thus the intensity of the attack on the host. The environment may also directly influence the host and therefore its response to attack. The age of the plant, the genotypes of host and pathogen and their interactions, microbial activity at the leaf surface are

all factors affecting the expression of resistance of a plant. These variables mean that direct comparisons of assessments of disease levels in plants as measured, for instance by the number of lesions per plant, the percentage area of infected,and/or destroyed leaf tissue or the percentage infected plants per pot are inappropriate. Biases may also be introduced by different observers. Large (1966) made several recommendations to counteract the effect of these variables and to relate disease assessment with yield response. He suggested that :

a) a close study of the morphology and physiological development of a healthy plant from sowing to harvest be made;
b) studies of disease development under a wide range of conditions in the field be made;
c) standard scales be used in assessing percentage plant tissue infected;
d) trials be run over many years and compared with control plots kept as free as possible from disease by spraying and management techniques.

Large suggested that these requirements be adapted to each disease situation to give uniform and comparable results. A decimal key for the assessment of the growth of a healthy host was introduced by Zadoks, Chang and Konzak (1974). Standard scales have been used in assessing the amount of disease on a plant since 1947, when the British Mycological Society produced a scale for assessing late blight of potatoes (*Phytophthora infestans*); Beaumont (1954) developed a scale for assessing damage caused by tomato leaf mould (*Cladosporium fulvum*) and N.I.A.B. have published scales commonly used today to assess mildew *(E. graminis)* and leaf blotch *(R. secalis)*.

Zadoks (1972) proposed the use of polycyclic and monocyclic tests to determine the resistance shown by a host plant. Polycyclic tests follow the progress of disease epidemics within a crop from the initial infection through increasing disease levels at recorded plant growth stages to harvest. Assessment is essentially at the macroscopic level. The behaviour of the plant in the field is the only true test of its resistance but experimentation in the field is not always practicable. The comparison of resistance shown by different plants is also unreliable as factors such as spore deposition and dissemination are not controllable. Glasshouse trials have the advantage that inoculation levels can be easily monitored and environmental conditions reproduced. These conditions, however, usually favour the development of the pathogen rather than the host and high levels of disease may mask small but important differences in resistance. Field and glasshouse trials can usefully be used as control for one another. The greater the number of cycles in one growing season, the more valid the results will be.

Monocyclic tests follow the development of a single unit of infection. Zadoks (1977) defined a unit of infection as "the mycelial structure that originated from a dispersal unit. It can be recognised visually, counted and/or measured up until the point that it, in turn, gives rise to a new dispersal unit." In the case of *E. graminis* and *R. secalis* the dispersal unit, a spore, becomes an infection unit as it germinates and goes through the stages of development viz: germ tube development; appressorial formation; penetration of the cuticle; haustorial formation and mycelial proliferation, until new conidia are formed which will re-infect the crop. Assessments at each stage of development are usually made microscopically.

The experiments which were undertaken in the present work were aimed to identify further sources of resistance to *E. graminis* and *R. secalis*, to assess the behaviour of the pathogen in response to resistance factors and to consider aspects of the uses of certain observed resistance factors in commercial practice.

EXPERIMENTAL STUDIES 1.

Assessments of barley lines and cultivars for resistance to mildew.

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Assessments of barley lines and cultivars for resistance to mildew.

Introduction.

The first series of studies was designed to screen a wide range of barley lines from different sources for resistance to infection by mildew. Lines of particular interest were those with high levels of resistance rather than those showing complete resistance which was thought to be a race specific response (Van der Plank, 1963). The barley lines used were taken from the European Barley Disease Nursery, the Expanded European Barley Disease Nursery and from the U.S.D.A. Small Grains Collection provided by Dr J.G. Moseman, U.S.D.A., Beltsville, Maryland. This last collection included entries from cultivated fields in Ethiopia, as well as wild examples of *Hordeum intermedium, H. vulgare, H. distichon, H. irregulare and H. deficiens,* lines of *H. vulgare* from Turkey and lines of *H. spontaneum* which had originated in Israel. A collection of commercial cultivars was also used. The study was carried out in three phases. The first (Experiment 1a) was concerned with the 700 lines in the collections exposed to natural sources of inoculum; the lines were visually assessed for mildew infection in three locations. From these assessments 69 lines which showed consistently slight mildew infection were selected. These 69 lines comprised the experimental material for the second stage of the study (Experiment 1b). The selected lines were grown under glasshouse conditions, in the same plots, over seven growth cycles; over the period they were again exposed to a natural mildew population. From the visual assessments made on these 69 lines, 13 lines selected as showing continuously high levels of resistance were assessed further for their reaction when challenged as leaf segments by different physiological races of *Erysiphe graminis*

representing a wide range of virulence factors (Experiment 1c). Microscopic observations on the development of *E. graminis* on inoculated leaves were also carried out in the final stage of this investigation.

Experiment 1a. Preliminary screening tests.

The preliminary screening programme was carried out on barley lines from the various collections (Appendix I) under three sets of conditions; i) in the field; ii) in an unheated glasshouse; iii) in a heated glasshouse. The aim of the study was to assess the lines for their response to mildew infection as a basis for the selection of sources of moderate to high levels of resistance.

Materials and methods.

In the field trial, seed was germinated in 2.5 cm fibre pots, filled with Levington seedling compost, in a glasshouse. The seedlings, still uninfected, were planted out in April 1977 at Growth Stage (G.S.) 12-13 (Zadoks, Chang, and Konzak, 1974) in field plots replicated three times. The plants were assessed for percentage mildew, chlorosis and necrosis at G.S. 40-50.

Lines in the unheated glasshouse were sown in pairs of rows 0.15 m apart with 0.5 m spacing between each pair of rows, in April 1977. Golden Promise was grown at the edge of each row and acted as a mildew source. Assessments of mildew, chlorosis and necrosis were made at 2-week intervals.

In the 15 m square heated glasshouse compartments, maintained at a temperature to sustain growth at all times, lines were again sown in pairs of rows with the same spacing as those in the unheated glasshouse. Golden Promise was planted at the edges of the rows and assessments were

made every 2 weeks.

Artificial inoculation was not necessary in any of the experiments, infection occurring naturally at the seedling stage (G.S. 10-20). The virulences present in the pathogen population were evidenced by the responses of the commercial cultivars with known resistances in Barley Mildew Resistance (B.M.R.) Groups R2 to R6 (Wolfe and Wright, 1978) grown in or near each experimental plot.

The assessments of the plants were based on the area of the upper four leaves affected by mildew and were classified into the following categories:

0 - no infection

1 - not more than 1 per cent infection

2 - " " " 10 " " "

3 - " " " 25 " " "

4 - " " " 50 " " "

5 - more than 50 per cent infection.

Results.

Mildew developed rapidly in all tests, particularly in the heated glasshouse (Plate 1). Many lines showed some necrotic spotting ranging from a slight flecking to about 80 per cent of the leaf area affected (Plate 2). The results are summarized in Table 1 and given more fully in Appendix II. In general, disease levels were higher in the glasshouse than in the fields. Virulences were present in the pathogen population to overcome resistances present in the commercial cultivars grown in the field and unheated glasshouse. The results of the different lines were not always the same in all three tests but lines considered to be of further interest in the investigation were those which always showed less than 10 per cent infection; i.e. were never

Plate 1. Mildew development in glasshouse compartments (Golden Promise).

Plate 2. Necrotic flecking on barley lines in glasshouse compartments. (line from Hordeum spontaneum collection).

Table 1. Summary of results of mildew assessments in preliminary screening tests.

Assessment series	Number of lines	Percentage of lines in each infection category					
		0	1	2	3	4	5
				European Collection			
Field (August, 1977)	44	18	18	39	20	5	0
Unheated Glasshouse (July 1977)	44	20	0	16	45	16	3
Heated Glasshouse (July-Oct.,1976)	47	4	15	23	30	11	17
				Expanded European Collection			
Field (August, 1977)	101	10	35	34	14	5	2
Unheated Glasshouse (July,1977)	100	21	14	31	29	5	0
Heated Glasshouse (July-Oct.,1976)	122	6	11	8	32	21	22
				Ethiopian Collection			
Field (August, 1977)	220	3	32	41	18	5	1
Unheated Glasshouse (July,1977)	240	26	1	13	51	8	1
Heated Glasshouse (Mar.-May, 1977)	237	1	1	37	35	22	4
				Hordeum spontaneum Collection			
Unheated Glasshouse (July,1977)	108	40	0	19	25	15	1
Heated Glasshouse (Mar.-May, 1977)	68	77	0	16	6	0	1
				Turkish Collection			
Field (August, 1977)	243	1	4	38	26	24	7
Unheated Glasshouse (July,1977)	245	6	0	3	30	57	4
Heated Glasshouse (Aprl.-June,1977)	244	1	0	12	53	27	7
				Commercial Collection			
Field (August, 1977)	40	13	27	42	13	5	0
Unheated Glasshouse (July,1977)	33	9	0	15	52	24	0

in an infection category higher than 2 but were not always in categories 0 or 1, evidencing major gene resistance. The Turkish collection was found to contain a high proportion of susceptible lines and only five were thought to be of further interest. Lines of *Hordeum spontaneum* were found to be generally resistant. Fifteen selections were made from this collection. Thirty lines from the Ethiopian collection were selected for further study as were three from the European and six from the Expanded European Barley Disease Nursery. The selections are those lines marked with an asterisk in Appendix I .

Experiment 1b. Further studies on various barley lines selected for low levels of mildew infection.

Lines selected from the results of the assessments in Experiment 1a as showing intermediate levels of resistance were grown in a heated glasshouse: in addition, 10 commercial barley cultivars with known resistance genes were sown to monitor the virulences present in the natural pathogen population. The various lines or cultivars were each grown repeatedly on the same site to promote selection within the pathogen population of any virulences to overcome particular resistance factors in the different lines. Seven growth cycles were studied between July 1978 and October 1979, but there was a gap in continuity between the third harvest and the fourth sowing.

Materials and methods.

Each plot consisted of a single plant of each line or cultivar grown in a 12.5 cm plastic pot. The experiment was laid out in a randomized block design with four replicates. Additional lighting was supplied by sodium vapour lamps and watering was carried out by an

Plate 3. Trickle irrigation system.

automatic trickle irrigation system (Plate 3). As plants approached maturity, pots containing new, uninfected seedlings were laid out in the same design as the first cycle so that seedlings and mature plants of the same cultivar or line were in close proximity. Thus the young plants were exposed to infection by any spores of *E. graminis* produced on an adult plant of the same line, to encourage the preferential selection of virulences for particular host genotypes.

Assessments of the percentage mildew on the four uppermost expanded leaves were carried out at 2-week intervals from the seedling stage to maturity.

Results.

Detailed assessments of each growth cycle are given in Appendix III and the relative responses of the lines and cultivars are summarized in Table 2. In the first growth cycle mildew levels were generally low. Of the commercial cultivars, Hassan (Mlas), Midas (Mla6), Zephyr (Mlg), Sonja (Mlh) and Golden Promise (no known major resistance genes) had the highest levels of infection, averaging 6 per cent to 8 per cent leaf area infected, followed by Vada (Mlv) and Wing (Mla4/7). Mazurka (Mlg + Mla4/7) and Maris Mink (Mlg + Mlas), both having more than one major gene for resistance, showed very little disease whilst Tyra (Mla) was uninfected. One line of those selected from the European Barley Disease Nursery was severely affected at the last two assessment dates of this growth cycle. Lines of the Expanded European Collection showed little or no infection over the four assessments. Within the Ethiopian Collection nine of the 38 lines showed levels of infection comparable with the more susceptible commercial cultivars at one or two of the assessment dates. Lines from *Hordeum spontaneum* were mainly

Table 2. Percentage lines in each collection with less than mean levels of mildew.

Cycle No.:	1				2			3		4	5			6				7	
Assessment Date:	14.6.78	20.6.78	29.6.78	11.7.78	7.9.78	21.9.78	8.10.78	21.10.78	13.12.78	28.3.79	3.5.79	16.5.79	24.5.79	4.7.79	11.7.79	19.7.79	3.8.79	10.9.79	3.10.79
Mean GS:	32	33	40	51	22	28	41	22	31	36	29	32	39	27	35	39	50	28	31
Mean Mildew: (%)	2	1	3	2	7	4	2	10	5	0.1	3	2	2	7	7	8	6	8	8
Expanded European	100	70	100	70	100	80	100	80	80	100	60	90	70	70	70	90	60	60	40
European	60	30	60	60	70	70	100	66	100	100	66	66	100	100	60	100	30	30	30
Ethiopian	60	63	61	60	46	60	58	40	50	97	63	52	52	41	66	43	39	41	43
Hordeum Spontaneum	90	90	89	90	100	87	87	60	65	90	85	80	90	56	53	53	48	40	27
Turkish	40	40	40	55	20	40	40	40	60	80	40	40	40	40	40	40	20	20	40
Commercial	40	50	40	10	20	10	30	10	20	100	30	20	30	20	10	10	20	10	10

resistant, although at one assessment two lines were more severely infected. Out of the five lines from the Turkish Collection, three had relatively high levels of mildew.

In the second growth cycle (Table 2) levels of mildew were generally greater than they had been at the beginning of the first cycle but declined as plants matured. All of the commercial cultivars showed some infection and mildew was particularly severe on Hassan, Midas, Golden Promise, Zephyr, Vada and Sonja. With one exception lines from the European and Expanded European Collections were infected only at a low level, as they had been in the first cycle. Six lines from the Ethiopian Collection showed amounts of disease in the susceptible range. None of the *Hordeum spontaneum* lines was heavily infected; three Turkish lines showed high disease levels.

At the third cycle (Table 2) levels of disease were high initially, with all commercial cultivars showing substantial levels of infection. One line of the European Collection was again more infected than the others and one line of the Expanded European Collection showed 10 per cent infection although the mean for this collection was 8 per cent. Twenty-six lines from the Ethiopian Collection showed levels of infection more than 10 per cent but 17 of these compared favourably with most commercial cultivars and were below 15 per cent infected. Six of the *H. spontaneum* lines reached 10 per cent, the remainder being only slightly infected and three lines from the Turkish Collection again showed a susceptible response with levels of infection between 12 per cent and 24 per cent.

Between the third harvest and the fourth sowing the glasshouse was cleared of plants and there were generally low levels of mildew in the fourth cycle (Table 2) which was planted in the same arrangement as the

previous three cycles.

From the fifth cycle, mildew levels increased (Table 2). The commercial cultivars gave the greatest average amount of mildew: Golden Promise and Midas were among the most severely infected. Levels of disease were low on all lines of the European and Expanded European Collections but eight lines of the Ethiopian Collection were more severely affected on at least one of the three assessment dates. One line of *Hordeum spontaneum* and two from the Turkish Collection appeared susceptible at this fifth cycle.

More mildew was present at the beginning of the sixth cycle than previously (Table 2) and all the commercial cultivars were again substantially infected. However, none of the European or Expanded European Collections showed disease levels in the susceptible range. Seventeen lines of the Ethiopian Collection were relatively severely infected in at least one assessment, as were three *H. spontaneum* lines and four Turkish lines.

In the seventh cycle (Table 2) disease levels were generally high and the commercial cultivars were usually severely infected, although low disease ratings were recorded for Vada at the first assessment and Sonja at the second. Two lines from the European Collection and one from The Expanded European Collection were substantially affected. The Ethiopian Collection revealed ten susceptible lines, the *H. spontaneum* collection one and the Turkish Collection four.

Table 3 indicates the growth cycles when each line showed a more than average (over all lines assessed) amount of mildew infection.

It may be seen that line 2005 from the European Collection showed always below average infection; four of the six Expanded European Collection and 17 of the 38 Ethiopian lines were similarly less

Table 3. Cycle (number given in brackets) in which each line or cultivar was classified as being relatively susceptible.

European Collection
2005
2015 (1,6,7)
2017 (7)

Expanded European Collection
1019
1021 (7)
1033
1057 (3)
1080
1101

Ethiopian Collection
3002 (6,7)
3003
3004
3008 (1,6)
3009
3013
3015 (5,7)
3018 (1)
3019
3024 (6,7)
3043 (3)
3044 (3)
3052 (3,7)
3055 (1)
3062
3065 (6)
3066 (6)
3067 (6)
3075
3086
3090 (6)
3091
3098 (1,6)
3103
3115 (2,6)
3122 (6,7)
3124
3125
3126 (7)
3142 (7)
3148
3182
3192 (1)
3210 (7)
3224
3225
3230 (3)
3242

H. spontaneum Collection
4033
4034
4051
4054 (3)
4066 (1,6)
4072
4084 (7)
4085
4090
4091
4092
4093 (6)
4094 (6)
4101 (1,3)
4114 (6)

Turkish Collection
5006 (6)
5010 (1,2)
5023 (1,3,5)
5135
5153 (1,6)

Commercial Cultivars
Hassan (3,6)
Midas (1,3,7)
Golden Promise (2,3,7)
Mazurka (3,6,7)
Wing (3,6)
Zephyr (1,2,3,7)
Vada (3)
Maris Mink (7)
Tyra (6)
Sonja (1,2)

infected in all cycles. In contrast most of the lines from the Turkish Collection, and cultivars from the Commercial Collection were consistently above average levels of infection.

A further illustration of the behaviour of the different lines and cultivars is given in Figure 6. The relative levels of mildew on each line or cultivar at the booting stage (G.S.44) for each growth cycle are given, confirming the trends towards resistance or susceptibility discussed previously.

Experiment 1c. Studies on selected lines of barley using isolates of *Erysiphe graminis* with known virulence genes.

Introduction.

Barley lines which had shown consistent resistance responses in experiment 1b were tested further using isolates of *E. graminis* with different characteristics. Commercial cultivars with known resistance factors were also included in the study as standard comparisons.

Materials and methods.

Thirteen lines of barley selected from the previous experiment and 11 commercial cultivars were grown initially in a spore-free environment in an isolation propagator based on a design of Jenkyn, Hirst and King, (1973) (Plates 4 and 5). Using a laminar flow cabinet, standard 2.5 cm segments were taken from the first and second true leaves of the seedlings when they were 16-17 days old. Segments of each line or cultivar were allocated to 10x10 cm transparent plastic petri dishes and arranged at random on water agar containing 80 ppm benzimidazole. This technique delayed senescence of the leaf tissue and results obtained have been found to correlate well with those from

Figure 6. The percentage mildew (transformed values) at approx.G.S.40 on lines and cultivars in each growth cycle 1-7.

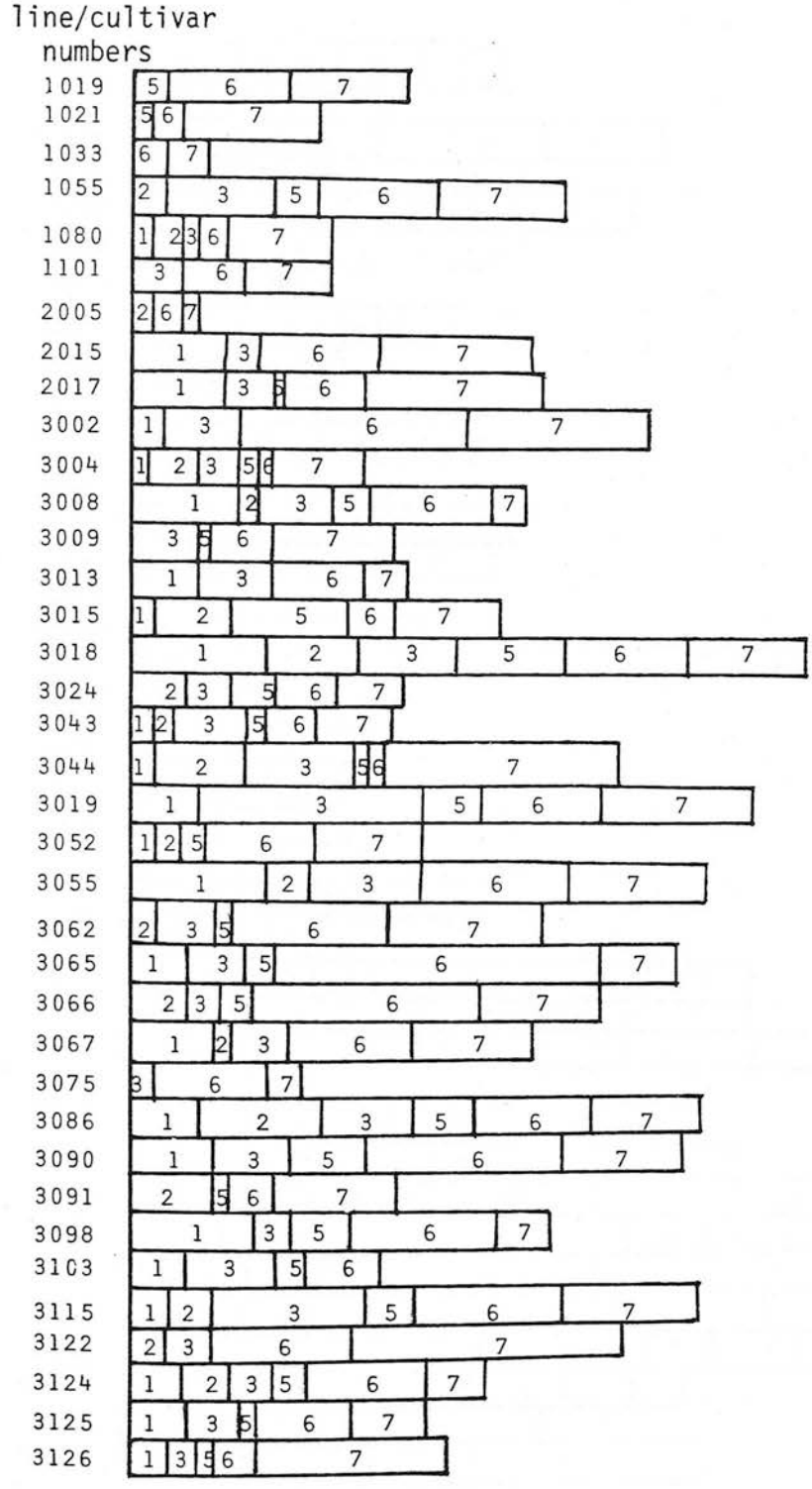


Figure 6.(continued)

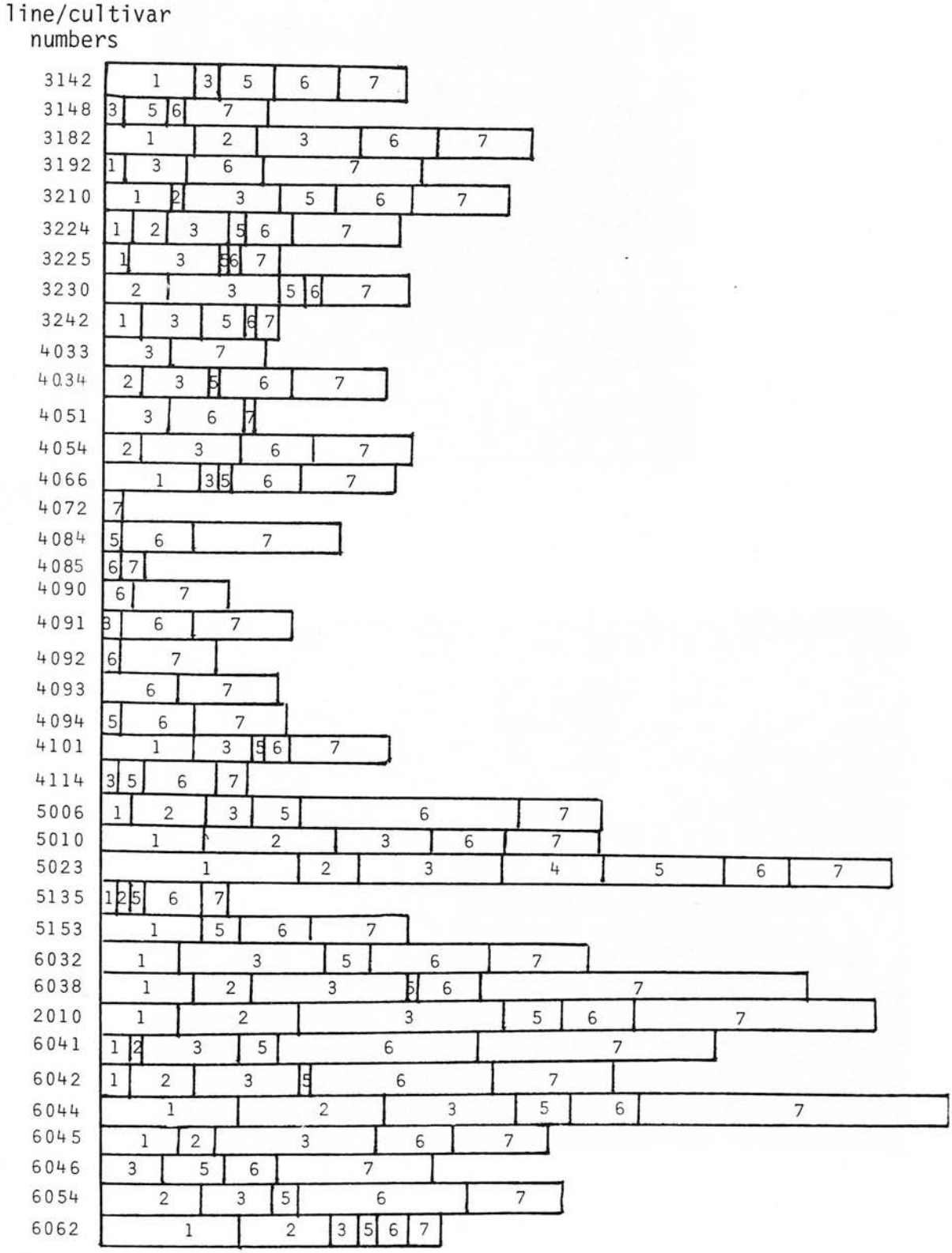


Plate 4. Isolation propagator.

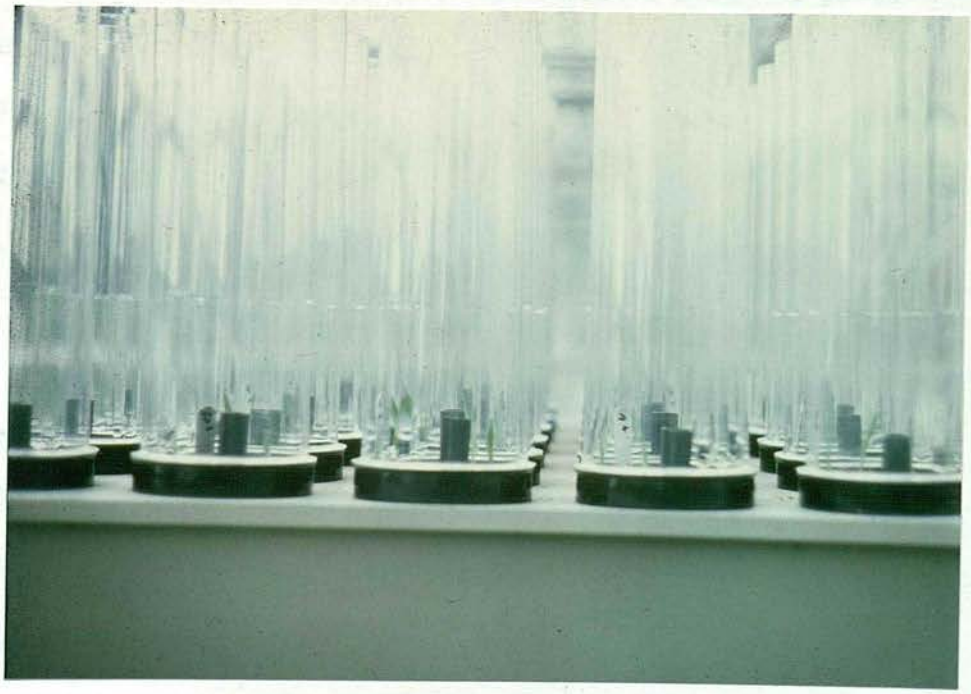


Plate 5. Isolation propagator.

field experiments (Jones and Hayes, 1971).

The experiment was arranged in a randomized block lay-out using a split-plot design with isolates forming the main plots and cultivars the sub-plots. There were four replicates.

Twelve isolates of *E. graminis* with known virulences were obtained from Dr. M.S. Wolfe, P.B.I., Cambridge and maintained on leaf segments of Golden Promise supported on benzimidazole agar. The conidia were inoculated with a camel-hair brush (Ouchi, Oku and Hibino, 1976) which was found to give a reproducible spore load in contrast to the "rolling method" of Masri and Ellingboe (1965), which gave an uneven spore distribution. The inoculated segments were incubated at 18-20°C in illuminated cabinets. Assessments of leaf area infected were made 7 days after inoculation using the scale of Jones and Hayes (1971). Assessments were also made of the extent of chlorosis and necrosis.

After visual assessments had been made the leaf segments were cleared overnight in Carnoy's solution (1 part acetic acid: 2 parts absolute alcohol) and stained in lactophenol cotton blue. After mounting in lactophenol the segments were assessed microscopically as to the extent of fungal development with different host/pathogen combinations.

Results.

From the analysis of variance of the transformed data (angles), levels of mildew were found to vary significantly with isolate and cultivar and there was a significant interaction between these two factors.

A summary of the mildew infection caused by each isolate averaged for all barley lines and cultivars is given in Table 4. Isolate 35

Table 4. Mildew development on leaf segments in relation to isolates of Erysiphe graminis (means of all lines and cultivars).

Isolate Number	Mildew	Percentage chlorosis	necrosis
	(transformed values)		
2	4.5	3.2	2.6
3	3.6	4.2	2.9
9	11.1	7.7	3.1
13	7.0	4.7	4.0
18	7.4	5.0	4.5
23	5.4	2.6	2.5
26	9.0	5.0	3.2
28	2.7	2.1	2.1
29	8.3	5.1	4.9
30	9.2	6.1	5.5
35	1.2	1.0	1.5
40	5.8	4.9	4.0
S.E.D.±	1.7	1.1	1.1

(D.F. = 33)

(V1,2) caused least infection while isolate 9 (V1,2,6,8) caused most.

The mildew infection level for lines and cultivars averaged over all isolates is given in Table 5. Of the commercial cultivars most showed above average levels of infection: exceptions were Midas, Tyra and Triumph with generally low infection rated, while Maris Mink and Simon had average levels of infection. Apart from Nos. 3075, 3091 and 1080, lines from the various collections showed below average infection levels.

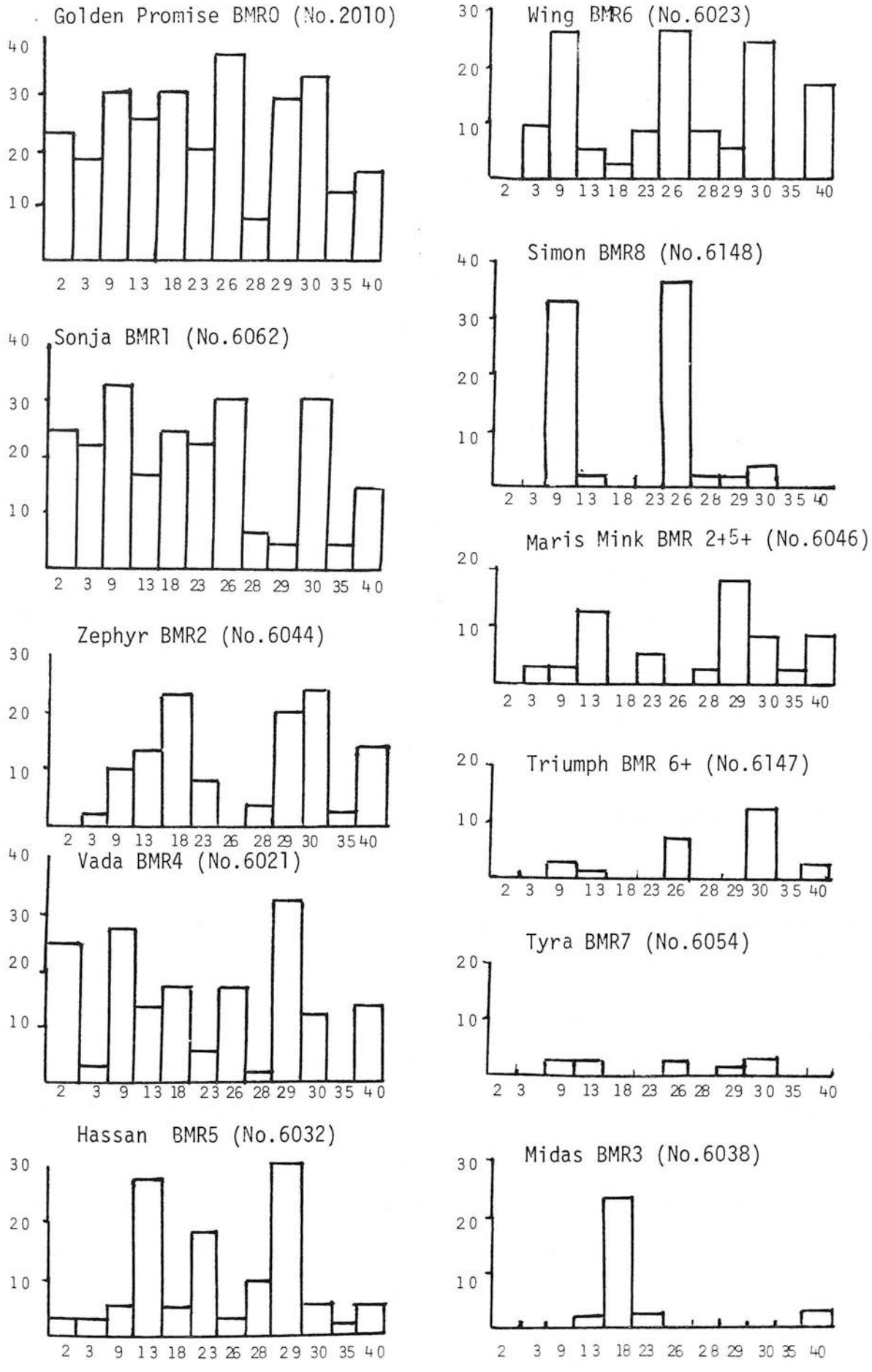
In considering the interaction between isolates and cultivars, the results for the commercial cultivars are summarized in Figure 7. Isolates 28 (V1,2,6) and 35 (V2) gave low rates of infection on all cultivars. Golden Promise (BMR0) and Sonja (BMR1) both showed similar patterns of response to all isolates apart from the low disease rating of isolate 29 (V1,2,4,5) on Sonja. Other cultivars showed a variable response to the different isolates. Zephyr (BMR2) had high disease levels (above 20 per cent) with isolates 18 (V1,5,7), 29 (V1,2,4,5) and 30 (V1,2,8) but low levels with isolates 2 (V1,4), 3 (V1,6) and 26 (V1,6,8). Vada (BMR 4) tended to high levels of infection with isolates 2 (V1,4), 9 (B1,2,6,8) and 29 (V1,2,4,5) but was more resistant to isolate 3 (V1,6). Hassan (BMR5) was most infected with isolates 13 (V1,2,5) and 29 (V1,2,4,5); very low levels of infection were favoured by isolates 2 (V1,4), 3 (V1,6), 9 (V1,2,6,8), 18(V1,5,7), 26 (V1,6,8) and 30 (V1,2,8). Wing (BMR6) gave a suceptible response with isolates 9 (V1,2,6,8), 26 (V1,6,8) and 30 (V1,2,8) but was resistant to isolates 2 (V1,4), 18 (V1,5,7) and 29 (V1,2,4,5). Midas (BMR3) was only infected substantially by isolate 18 (V1,5,7), Maris Mink (BMR 2+5+) by isolates 13 (V1,2,5) and 29 (V1,2,4,5) and Simon (BMR8) by isolates 9 (V1,2,6,8) and 26 (V1,6,8). Tyra (BMR7) showed consistently low levels of infection as did Triumph

Table 5. Mildew development on leaf segments in relation to barley line or cultivar. (Means of all isolates).

Line or cultivar	mildew	Percentage chlorosis (transformed values)	necrosis
1033	2.5	2.1	3.9
3075	7.1	6.4	3.7
4085	2.0	4.0	3.8
4092	1.0	1.2	2.3
3224	1.3	1.5	3.8
3055	4.6	4.1	3.9
2005	2.4	2.5	2.5
4090	1.9	1.8	3.3
3091	7.2	6.2	2.9
1080	8.8	7.8	5.0
1021	2.6	5.1	3.0
4072	1.2	2.5	2.0
3230	1.1	1.3	3.9
Golden Promise	22.9	7.6	3.5
Sonja	21.3	5.5	3.3
Zephyr	11.0	5.8	2.3
Midas	2.3	1.7	2.2
Vada	14.7	8.0	5.5
Hassan	9.4	6.3	5.4
Wing	10.8	5.9	5.9
Tyra	0.8	3.3	1.3
Simon	6.5	5.4	1.7
Maris Mink	5.1	4.1	3.6
Triumph	1.9	2.8	3.1
S.E.D.±	1.3	1.1	0.8

(D.F. = 828)

Figure 7. The percentage mildew on barley cultivars for each isolate. SED I



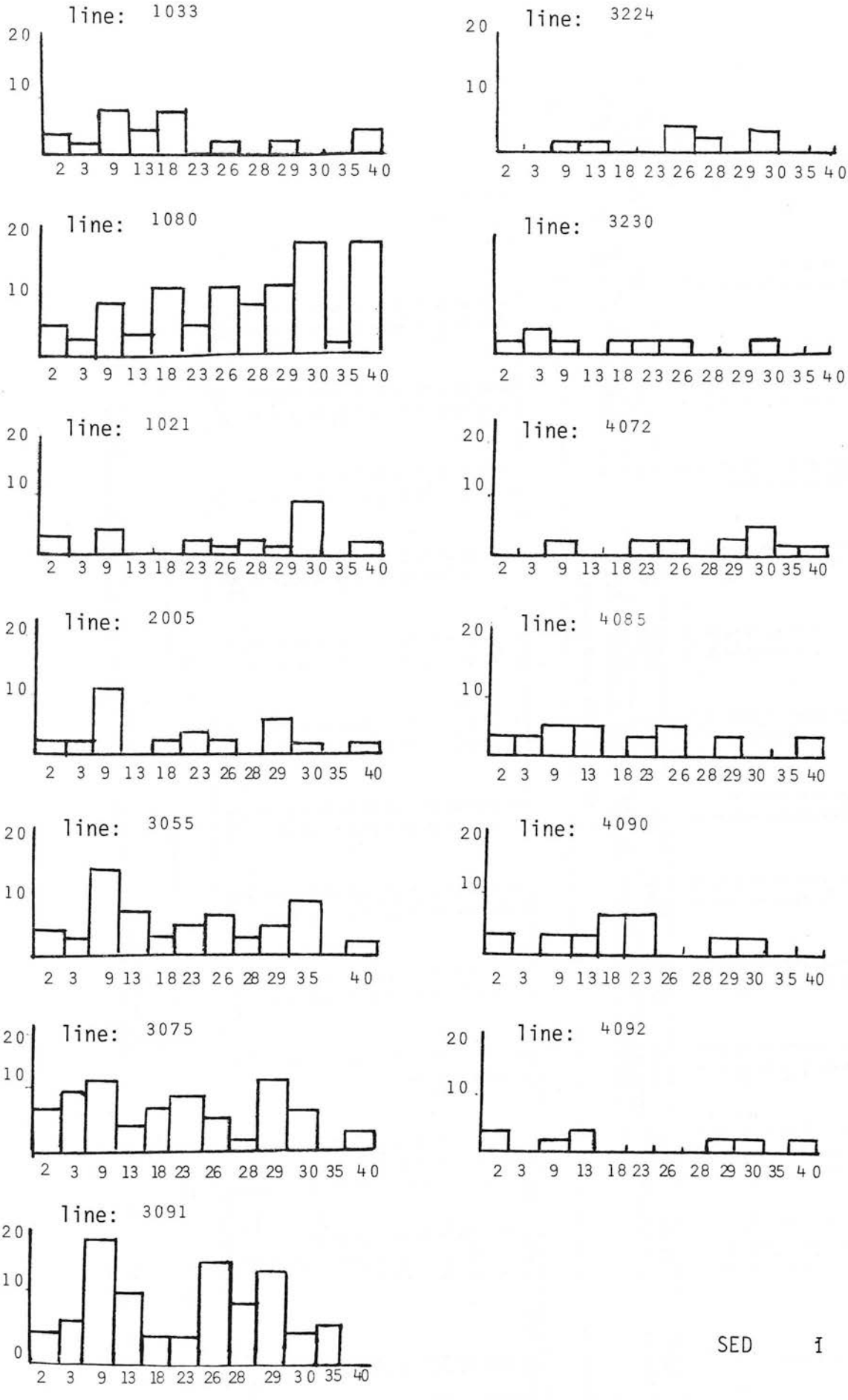
(BMR6+) apart from moderate infection with isolates 26 (V1,6,8) and 30 (V1,2,8).

The results of interactions between isolates and barley lines are given in Figure 8. No lines showed over 20 per cent infection on average and five of the lines were consistently below 5 per cent infected. Intermediate levels of resistance usually were found with lines 3075 and 3055. More variable responses to isolates were shown by lines 3091, 1080, 2005 whilst lines 4090, 1021 and 1033 showed intermediate or low levels of disease.

The levels of chlorosis and necrosis which developed in relation to isolate and line or cultivar are indicated in Tables 6 and 7. Significant differences were associated with some isolates and line or cultivar combinations although there were no interactions between the two factors. Levels of chlorosis were associated generally with levels of mildew although the most susceptible cultivars were not the most chlorotic. The incidence of necrosis was related to mildew levels with respect to isolate, although the differences were generally small. With respect to line or cultivar, No. 1080, Vada, Hassan and Wing were most necrotic.

Microscopic assessments are summarized in Table 8. Golden Promise with no known major genes for resistance was extensively colonized by all isolates of *E. graminis* although mycelium formation and spore production was less prolific with isolate 18 (V1,5,7) than any others. Sonja, Vada and Hassan also exhibited a susceptible response with all isolates. In contrast lines 4085 and 4072, both of which had less than average amounts of mildew when visually assessed, were shown at the microscopic level to give a moderately resistant or moderately susceptible response to most pathogen isolates.

Figure 8. The percentage mildew on barley lines for each isolate.



SED 1

Table 6. Percentage chlorosis (transformed values) of each isolate/line or cultivar interaction.

Isolate number	Virulence factor	Line number											
		1033	3075	4088	4092	3224	3055	2005	4090	3091	1080	1021	4072
2	1,4	1.4	4.3	4.3	1.4	1.4	2.9	1.4	1.4	0.0	5.7	5.7	2.9
3	1,6	2.9	8.9	5.7	0.0	2.9	2.9	4.3	2.9	2.9	2.8	6.1	5.7
9	1,2,6,8	2.9	9.3	6.1	0.0	0.0	6.1	4.6	1.4	15.7	10.6	1.4	5.7
13	1,2,5	2.9	5.7	5.7	2.9	1.4	2.9	2.9	1.4	9.7	4.3	4.3	2.9
18	1,5,7	6.1	7.5	1.4	0.0	0.0	2.9	2.9	4.3	2.9	10.7	1.4	2.9
23	1,5	0.0	11.1	4.8	2.9	0.0	2.9	0.0	1.4	2.9	4.3	6.1	1.4
26	1,6,8	2.9	7.5	7.5	0.0	2.9	4.3	2.9	1.4	15.9	9.3	6.1	0.0
28	1,2,6	0.0	0.0	2.9	2.9	0.0	1.4	0.0	0.0	4.6	6.1	4.7	1.4
29	1,2,4,5	2.9	10.7	4.7	0.0	0.0	5.7	6.0	2.9	9.3	7.8	2.9	1.4
30	1,2,8	0.0	3.2	1.4	2.9	7.8	11.5	1.4	3.2	2.9	14.7	14.4	2.9
35	1,2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.6	1.4	1.4	1.4
40	1,3,4	2.9	7.9	5.7	1.4	1.4	5.7	2.9	1.4	2.9	15.9	6.1	1.4
mean SED±1.14(DF=33)		2.1	6.3	3.9	1.2	1.5	4.1	2.5	1.8	6.2	7.8	5.1	2.5

Isolate number	Virulence factor	Line/cultivar name/number												Mean of all cultivars/lines SED±1.1(DF=828)
			G.Promise	Sonja	Zephyr	Midas	Vada	Hassan	Wing	Tyra	Simon	Maris Mink	Triumph	
		3230	2010	6062	6044	6038	6021	6032	6023	6054	6148	6046	6147	
2	1,4	0.0	3.2	2.9	4.3	3.2	9.3	2.9	6.1	1.4	1.4	4.3	4.3	3.2
3	1,6	4.3	6.0	7.9	7.5	0.0	2.9	5.7	4.3	6.1	2.9	1.4	3.2	4.1
9	1,2,6,8	1,4	24.9	16.6	7.5	0.0	20.8	7.5	16.1	4.7	12.9	4.3	4.3	7.7
13	1,2,5	1.4	4.7	2.9	7.9	2.9	14.1	9.3	4.3	4.3	5.7	4.3	2.9	4.7
18	1,5,7	4.3	16.6	12.9	1.4	8.3	10.7	2.9	1.4	3.2	6.0	7.5	1.4	5.0
23	1,5	0.0	0.0	0.0	6.1	0.0	1.4	1.4	4.7	4.6	1.4	1.4	2.9	2.6
26	1,6,8	0.0	6.7	3.2	1.4	4.7	6.0	6.1	11.5	1.4	14.4	1.4	1.4	4.9
28	1,2,6	1.4	4.3	4.7	1.4	0.0	1.4	6.0	3.2	0.0	4.7	1.4	0.0	2.1
29	1,2,4,5	0.0	4.7	1.4	7.8	0.0	12.5	19.8	6.0	2.9	1.4	9.3	2.9	5.1
30	1,2,8	1.4	8.9	6.1	14.4	1.4	6.5	7.5	6.0	6.5	7.9	6.1	6.5	6.1
35	1,2	0.0	1.4	4.3	4.3	0.0	0.0	1.4	1.4	0.0	0.0	0.0	1.4	0.1
40	1,3,4	1.4	9.3	2.9	5.7	0.0	10.7	4.3	6.0	4.6	6.0	7.9	2.9	4.9
mean SED±1.14(DF=33)		1.3	7.5	5.5	5.8	1.7	8.0	6.3	5.9	3.3	5.4	4.1	2.8	

Table 7. Percentage necrosis (transformed values) of each isolate/line or cultivar interaction.

Isolate number	Virulence factor	Line number											
		1033	3075	4088	4092	3224	3055	2005	4090	3091	1080	1021	4072
2	1,4	4.3	4.3	1.4	1.4	2.8	1.4	2.8	2.8	0.0	5.7	2.8	2.8
3	1,6	4.3	2.8	2.8	2.8	2.8	2.8	2.8	2.8	4.3	2.8	1.4	4.3
9	1,2,6,8	2.8	1.4	6.0	0.0	5.7	4.6	2.8	7.5	1.4	4.3	2.8	4.3
13	1,2,5	4.3	4.3	4.6	4.3	2.8	4.6	2.8	2.8	2.8	2.8	4.3	2.8
18	1,5,7	4.3	4.3	3.2	1.4	4.3	4.6	1.4	5.7	2.8	4.6	4.3	1.4
23	1,5	2.8	2.8	1.4	4.3	1.4	4.3	2.8	4.3	2.8	6.1	1.4	0.0
26	1,6,8	1.4	4.3	9.3	1.4	2.8	2.8	2.8	1.4	6.0	7.4	2.8	2.8
28	1,2,6	1.4	0.0	0.0	1.4	6.4	4.4	1.4	0.0	0.0	6.0	1.4	0.0
29	1,2,4,5	5.7	8.9	7.5	0.0	5.7	5.7	2.8	4.3	2.8	6.4	2.8	0.0
30	1,2,8	8.9	6.4	6.0	4.4	4.6	6.1	4.3	4.6	6.1	4.6	4.3	2.8
35	1,2	2.8	1.4	0.0	1.4	0.0	0.0	2.8	0.0	1.4	1.4	2.8	1.4
40	1,3,4	2.8	2.8	2.8	4.3	5.7	7.5	0.0	2.8	4.3	7.5	4.3	1.4
mean	SED±1.08(DF=33)	3.9	3.7	3.8	2.3	3.8	3.9	2.5	3.3	2.9	5.0	3.0	2.0

Isolate number	Virulence factor	Line/cultivar name/number												Mean of all cultivars SED±0.81(DF=828)
			G.Promise	Sonja	Zephyr	Midas	Vada	Hassan	Wing	Tyra	Simon	Maris Mink	Triumph	
		3230	2010	6062	6044	6038	6021	6023	6023	6054	6148	6046	6147	
2	1,4	4.3	0.0	0.0	1.4	0.0	1.4	7.5	7.9	1.4	2.8	1.4	0.0	2.6
3	1,6	7.5	1.4	2.8	0.0	1.4	6.4	6.1	2.8	1.4	0.0	0.0	3.2	2.9
9	1,2,6,8	4.3	0.0	1.4	1.4	1.4	6.0	7.4	1.4	0.0	0.0	2.8	4.3	3.1
13	1,2,5	4.6	7.8	2.8	4.6	1.4	4.6	4.6	7.5	2.8	2.8	4.3	4.3	4.0
18	1,5,7	6.1	9.2	6.0	1.4	1.4	16.2	5.7	9.6	1.4	1.4	6.1	1.4	4.5
23	1,5	1.4	0.0	0.0	1.4	4.6	2.8	1.4	9.3	0.0	1.4	1.4	1.4	2.5
26	1,6,8	0.0	0.0	1.4	0.0	0.0	6.0	7.5	3.2	2.8	3.2	2.8	3.2	3.2
28	1,2,6	4.6	2.8	4.6	1.4	5.7	2.8	3.2	0.0	0.0	1.4	2.8	0.0	2.1
29	1,2,4,5	1.4	3.2	6.1	3.2	4.6	6.1	4.6	16.2	1.4	3.2	9.3	5.7	4.9
30	1,2,8	6.4	9.2	9.6	2.8	1.4	6.0	9.3	6.1	2.8	4.3	4.3	6.1	5.5
35	1,2	2.8	4.6	2.8	4.3	1.4	0.0	1.4	1.4	0.0	0.0	1.4	0.0	1.5
40	1,3,4	2.8	2.8	1.4	5.7	2.8	7.5	6.1	4.6	1.4	0.0	6.1	7.5	4.0
mean	SED±1.08(DF=33)	3.9	3.5	3.3	2.3	2.2	5.5	5.4	5.9	1.3	1.7	3.6	3.1	

Table 8. Microscopic evaluation of Erysiphe graminis development in different host/pathogen combinations.

Isolate Number	Virulence Factor	Line or cultivar number, name and resistance group																						
															Golden Promise	Sonja	Zephyr	Midas	Vada	Hassan	Wing	Tyra	Simon	Maris Mink	Triumph
															0	1	2	3	4	5	6	7	8	2+5+	6+
		1033	3075	4085	4092	3224	3055	2005	4090	3091	1080	1021	4072	3230	2010	6062	6044	6038	6021	6032	6023	6054	6148	6046	6147
2	1,4	s	S	s	r	s	S	s	s	s	s	s	s	s	S	S	s	s	S	S	s	R	s	s	l
3	1,6	r	S	s	s	R	s	R	R	s	S	s	s	S	S	S	S	s	S	S	S	S	R	R	R
9	1,2,6,8	s	S	s	r	s	S	S	s	S	s	S	s	S	S	S	S	S	S	S	S	R	R	s	s
13	1,2,5	S	s	s	l	r	s	r	s	s	S	S	s	R	S	S	S	s	S	s	s	r	S	S	s
18	1,5,7	s	S	S	s	R	S	S	s	S	S	l	r	S	s	S	s	S	S	s	S	R	R	s	l
23	1,5	s	S	s	s	R	S	S	r	s	s	s	r	s	S	S	S	S	S	S	S	r	S	S	s
26	1,6,8	R	S	s	r	S	S	S	R	S	s	S	r	s	S	S	R	R	S	S	S	r	R	l	s
28	1,2,6	R	S	s	s	s	S	l	l	s	S	l	l	R	S	s	s	s	S	S	S	r	R	S	s
29	1,2,4,5	s	S	s	S	s	S	S	S	S	S	l	R	S	S	s	S	s	S	S	S	s	S	S	s
30	1,2,8	r	s	s	s	s	S	S	R	S	S	S	s	s	S	S	S	s	S	S	S	R	R	S	s
35	1,2	l	s	l	l	l	l	l	l	S	s	l	l	R	S	l	r	l	l	S	r	l	l	l	s
40	1,3,4	s	S	r	s	s	s	R	R	S	S	s	R	R	S	S	s	R	S	S	S	r	l	S	S

S = susceptible; prolific mycelium, spore production
s = moderately susceptible; sparse mycelium, limited spore production
r = moderately resistant; sparse mycelium, no spore production
R = resistant, no development beyond appressorium

l - not recorded

DISCUSSION

In the preliminary screening tests based on field and glasshouse trials, a wide range of response to infection by a natural inoculum was shown by the lines and cultivars of the different barley collections. Precise assessments of the behaviour of individual lines were not expected from the first investigations which were simply intended to place the large number of entries into broad groups of plants showing similar resistance responses, as a basis for selection. Mildew infection occurred in all the trials but overall levels were lower in the field trials than the glasshouse, where conditions proved very favourable for the pathogen. This may have led to a masking of some types of partial resistance which could have proved effective in the field.

In considering the preliminary results for the various collections, the Turkish Collection contained the largest proportion of susceptible lines. In a previous study on material from the U.S.D.A. Small Grains Collection, of which the Turkish Collection formed a part, Caddel (1976), working in Morocco, found that entries from Turkey occupied a middle position with regard to resistance. The differences in the findings of the two studies may relate to variations in virulence factors associated with the natural pathogen populations used. In the present work the responses of commercial cultivars with known resistances indicated that plants were exposed to a wide range of virulence factors for the duration of the trials. The Collections showing the greater numbers of highly resistant lines were the commercial, European and Expanded European groups, all of which were known to provide a range of major gene resistances (Wiberg, 1974; Wolfe *et al*, 1980). The *Hordeum spontaneum* entries collected in Israel and known

to have resistance to leaf rust or mildew or both also encompassed many lines which proved to be highly resistant. The Ethiopian Collection, comprising plants originally from cultivated fields in Ethiopia, also contained a large number of lines which showed intermediate levels of resistance. For the second stage of the screening programme, lines and cultivars which showed intermediate levels of resistance were chosen for further study and the majority of selections were from the Ethiopian Collection.

Interest in forms of resistance which act at an intermediate level is due to their apparent association with long-term effectiveness. According to Schwarzbach and Wolfe (1975), intermediate levels of resistance may be more durable than high levels as they exert less selective pressure towards corresponding virulences in the pathogen population. Russell (1978) observed that growing near-immune varieties of crop plants imposed a great selection pressure in favour of resistance-breaking variants of the parasite concerned, and the development of partially resistant varieties may be of more long-term value. There are, however, difficulties in distinguishing between durable and transient forms of resistance without exposing the resistance to the parasite for many years. Wolfe (1972) indicated that incomplete or partial resistance may also be non-specific but this is difficult to assess.

Further screening for partial resistance in the second stage of these investigations, based on selections made from preliminary studies, was based on exposing plants continuously to inoculum from a previous generation of the same host in conditions favourable for fungal development. In this way, it was hoped to intensify the selection of host-specific genotypes of the pathogen. From the first to the second

crop cycles there was an increase in the levels of mildew, partly attributable to a build-up of the general inoculum but further increase may also reflect the increase in prevalence of particular virulence factors within the fungal population to overcome resistance factors in the cultivars or lines concerned. Resistance in the commercial cultivars based on the genes Mlh, Mlg, Mla6, Mlv, Mlas and Mla4/7 was overcome and the time when resistance was overcome (Table 3) may reflect the frequency of the virulences in the original, natural pathogen population. Thus, cultivars showing delayed susceptibility possessed less widely used or more recently introduced genes or had a combination of resistance factors. The behaviour of the selected lines varied considerably: some proved highly susceptible, particularly those from the Turkish Collection, perhaps reflecting inadequacies in the original trials, while others showed increasing levels of infection with later growth cycles at some growth stages. A third group showed consistently below average levels of infection which were selected for further study with known isolates of *Erysiphe graminis* representing a range of virulence factors.

In the third study, the second selections and some commercial cultivars were tested, using leaf segments inoculated in controlled conditions with known isolates of the fungus, previously shown to possess particular virulence factors. In some cases the expected responses of certain cultivar-isolate combinations did not occur, possibly due to some contamination of isolates: however, isolates mostly performed in a theoretically consistent way when tested against known cultivars. Variation in average levels of infection occurred with different isolates as well as with different cultivars and this may be partly related to the number or frequency of virulence factors

and the respective major resistance factors present. For example, isolates with greater numbers of virulence factors showed, on average, high levels of infection (Table 4) and cultivars compatible with the greatest number of isolates i.e. one or more virulence factor was present to overcome one or more resistance factors, also showed greater levels of infection. (Table 5).

Variation in the overall performance of isolates, however, was not completely explained by their virulence factors. For example, in comparing isolates 28(V1,2,6) and 30(V1,2,8) both with three virulence factors, the average level of mildew for isolate 30 was significantly higher. Similarly, with cultivars, variation in average disease ratings was not always a reflection of major resistance factors: for example Hassan showed lower disease levels than Vada , even though Hassan was exposed theoretically to a greater number of compatible isolates. The quantitative variation in the behaviour of isolates is also illustrated in comparing the response of cultivars susceptible to a number of different isolates. Golden Promise and Sonja, for instance, behaved very differently, although both were theoretically susceptible to all isolates. In general, the commercial cultivars showed vertical resistance responses to isolates (Van der Plank, 1963) and with compatible races infection levels were high: exceptions were Tyra and Triumph which did not conform to this pattern but were only tested with one or two compatible isolates. In comparison with the commercial cultivars, the selected lines showed a greater consistency of behaviour over the range of different isolates, suggesting horizontal resistance characteristics (Van der Plank, 1963). Five lines showed very low levels and two lines intermediate levels throughout; of the remaining lines, three usually showed intermediate or low

disease levels and three a more variable response; infection was not, however, high on any line. Thus, some lines may have demonstrated a source of non-race specific resistance at an intermediate level. When lines showed low levels of disease throughout, it is possible that major gene effects were operating and it may be noticed that none of the isolates tested was effective against Tyra (Mla), whose resistance is derived from the cultivar Algerian (Torp, Jensen and Jørgensen, 1978) and which had a response profile similar to that of the highly resistant barley lines.

The results of the microscopic assessments gave no clear indication of behaviour characteristics associated with different resistances. Most isolate/cultivar or line combinations gave some spore producing colonies even when the relationship was theoretically incompatible. More quantitative work would have been necessary to establish precisely the expression of resistance in terms of fungal behaviour.

EXPERIMENTAL STUDIES 2.

Studies on the development of *Erysiphe graminis* on commercial cultivars of barley with known resistance genotypes.

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Studies on the development of *Erysiphe graminis* on commercial cultivars of barley with known resistance genotypes.

Introduction.

In these studies various barley cultivars grown commercially, either in the past or at present, were investigated. In the first study (Experiment 2a) cultivars with no known major genes for resistance were assessed; of these only Golden Promise and Proctor are widely grown today. In the second study (Experiment 2b) cultivars with resistance gene Mlg, the first to be extensively used in commercial breeding programmes in Britain (Howard, Johnson, Russell and Wolfe, 1970) were grown with Maris Concord and Midas (Mla6) and Impala with resistance factors Mlg and Mla6. Two further studies (Experiment 2ci and 2cii) were concerned with a wide range of more recently introduced varieties with different resistance factors.

Experiment 2a. Mildew development on cultivars with no known major genes for resistance.

The majority of the cultivars used in this study are out-dated although several of them have previously occupied an important position in barley production. The aim of the experiment was to assess the amount of variation expressed by the various cultivars in response to mildew infection.

Materials and methods.

Fifteen spring barley cultivars were used in the study which covered two growth cycles. The experiment was carried out in the same glasshouse, at the same time and under the same conditions as Experiment 1b. In this experiment eight replicates were laid out in a randomized block design, each plant was grown in a 12.5 cm pot. Assessments were made at the early tillering stage (G.S. 20) and at stem elongation (G.S. 35) or early booting (G.S. 40).

Results.

The results are summarized in Table 9. In the first cycle the amount of disease present was generally small apart from a moderate level on Spratt at the first assessment. Later, infection became more severe especially in Freja, Plumage and Plumage Archer, as well as Spratt. Asse and Clermont were least affected at both assessments.

In the second growth cycle mildew levels were generally higher. Again Asse showed generally less infection than other cultivars.

Experiment 2b. Mildew development on barley cultivars with known major gene resistance.

It had been observed from Experiment 2a that even when no known major resistance factors were present, barley varieties exhibited varying resistance to mildew infection. This experiment was designed to determine whether cultivars with a major resistance would also display such variable responses when virulences present in a pathogen population overcame the major resistance. Cultivars with resistance conditioned by Mlg, Mla6 (Maris Concord and Midas) or with both Mlg and Mla6 (Impala) factors were considered.

Table 9. Percentage mildew (M), chlorosis (C) and necrosis (N) of cultivars with no known resistance genes (transformed values).

Cultivar	6 June (GS 26)			13 June (GS 40)			3 Aug (GS 30)			12 Aug (GS 34)		
	M	C	N	M	C	N	M	C	N	M	C	N
Archer	3	2	25	9	4	24	12	5	0	13	12	34
Asse	0	3	12	3	3	9	8	4	1	2	6	23
Clermont	1	4	22	5	5	12	6	3	0	11	8	32
Freegold	4	3	11	12	5	12	11	4	0	13	10	27
Freja	2	1	22	13	5	17	9	4	1	14	12	33
Golden Promise	0	2	15	10	5	8	14	7	0	16	12	28
Maythorpe	1	3	11	8	4	9	16	9	0	8	11	27
Nymphe	4	4	7	8	5	16	11	4	1	8	11	26
Pallas	4	2	29	12	5	11	8	4	0	10	9	41
Plumage	3	2	31	19	8	21	10	3	0	16	12	27
Plumage Archer	1	2	26	17	5	17	9	3	0	11	10	33
Proctor	2	2	32	12	5	13	8	3	0	15	9	30
Spratt	10	4	26	23	5	12	10	3	0	14	13	34
Spratt Archer	1	3	27	8	3	13	7	2	0	11	12	30
Ymer	5	5	22	12	7	13	11	6	1	13	13	29
S.E.D.±	2.2	1.7	4.9	3.1	1.5	4.6	1.6	0.9	0.3	2.4	2.3	4.8

Materials and methods.

The same procedures were used as in Experiment 2a, using fifteen further barley cultivars, some grown on a commercial scale and some which have been superseded by other cultivars.

Results.

The results are summarized in Table 10.

At the first assessment disease levels were low, generally less than 5% although Gerkra and Goldfoil were most highly infected. At the second assessment overall levels of mildew increased; C.P. 127422 was least infected followed by Armelle, Julia and Impala.

At the beginning of the second cycle, mildew levels were higher than previously; Gerkra and Goldfoil were again highly infected. At the final assessment C.P. 127422 and Armelle appeared the least infected.

Overall Armelle, Julia, Union and C.P. 127422 tended to be less diseased than other cultivars.

Experiment 2c. Assessment of mildew development on commercial cultivars possessing different resistance genes.

In 1978 a glasshouse study was carried out using cultivars of different BMR groups possessing different known genes for resistance to mildew. The pattern of mildew development throughout the growth of the plants was assessed along with the development of senescence.

Table 10. Percentage mildew (M), chlorosis (C) and necrosis (N) of cultivars with M1g, M1a6 or M1g + M1a6 resistance. (transformed values)

Cultivar	6 June (GS 21)			13 June (GS 39)			3 Aug (GS 30)			16 Aug (GS 34)		
	M	C	N	M	C	N	M	C	N	M	C	N
M1g												
Armelle	1	8	22	9	6	19	11	4	0	8	8	40
Berac	6	4	16	16	9	20	11	5	1	12	9	29
Cambrinus	4	2	19	17	5	12	8	3	1	16	12	29
Deba Abed	4	1	17	13	5	12	6	2	0	17	10	27
Gerkra	9	1	5	16	5	8	19	7	0	15	9	25
Goldfoil	9	2	20	16	5	11	19	9	0	14	11	35
Imber	3	3	13	11	6	12	8	3	0	16	9	27
Julia	3	2	12	11	5	22	9	3	0	13	10	24
Mosane	4	2	15	15	5	11	10	4	0	16	9	27
Union	1	1	19	12	6	12	7	3	0	15	10	29
Zephyr	4	2	18	14	5	7	15	6	1	16	14	28
CP 127422	1	2	17	7	5	12	16	7	0	11	10	31
M1a6												
Maris Concord	5	3	23	16	7	16	5	1	0	18	9	30
Midas	6	2	20	15	5	10	11	5	2	14	11	26
M1g+M1a6												
Impala	3	1	18	12	7	13	7	3	0	17	10	33
S.E.D.±	2.6	1.2	5.2	2.9	1.5	4.4	1.8	0.8	0.3	2.9	1.6	5.5

Materials and methods.

Thirty commercial cultivars of spring barley belonging to various barley mildew resistance groups were assessed (Table 11). The cultivars were sown on 7 May 1978 in an unheated glasshouse. Each plot consisted of approximately 10 plants in a 15 cm circle, supported by a wire ring on a cane. The spacing between plots was 15 cm, and six replicates were arranged in a randomized block design. Golden Promise was grown around the plots to provide a source of inoculum.

Assessments began on 15 June when plants had developed to about G.S. 30. The percentage area of the top four fully expanded leaves infected by mildew was determined using the A.D.A.S. Barley Mildew Key No. 111. The percentage leaf area affected by chlorosis and necrosis was also recorded. Six assessments were made at two-week intervals until the onset of ripening at G.S. 70 to 80.

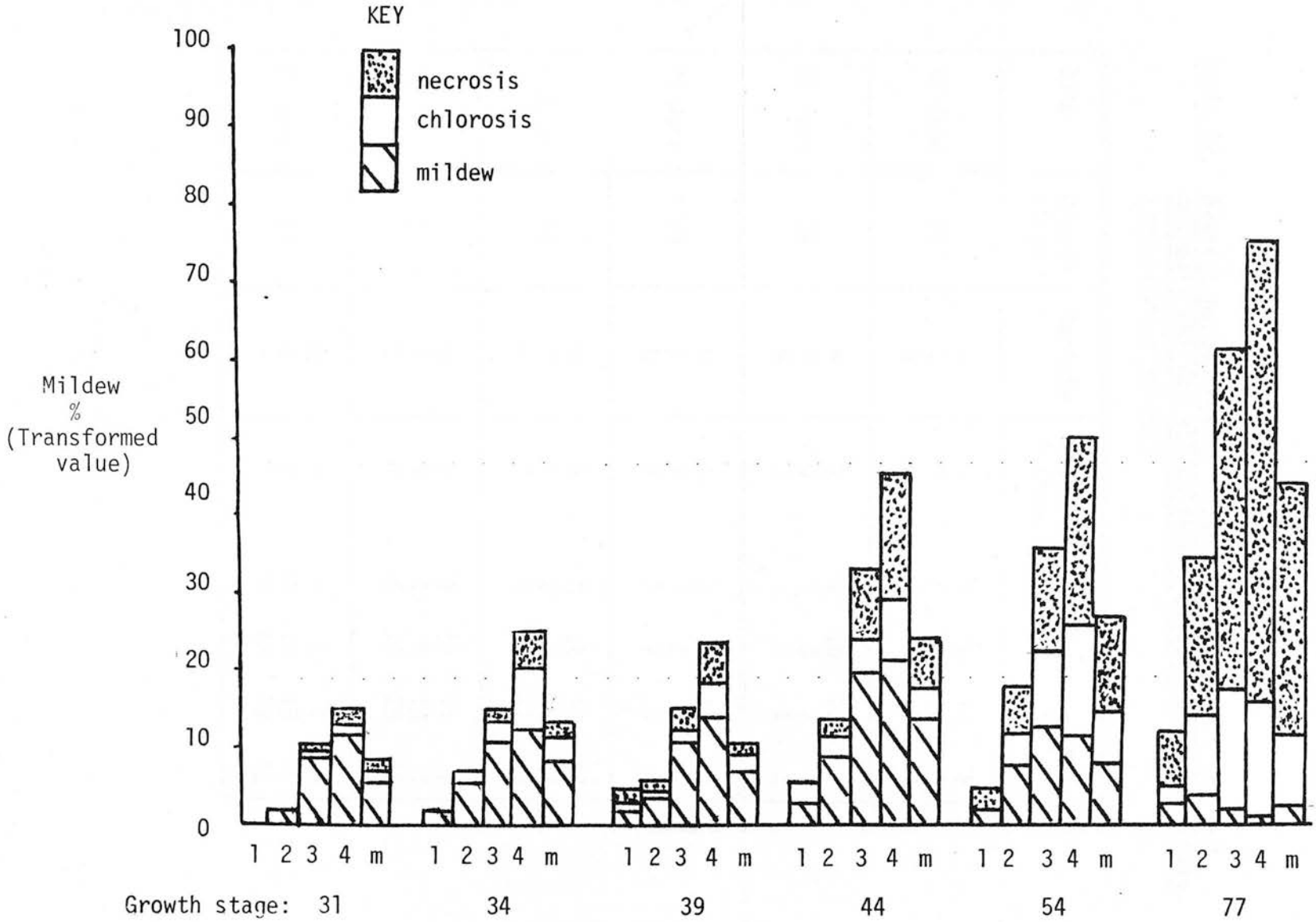
Results.

The average levels of mildew, chlorosis and necrosis for all cultivars at each assessment are given in Table 12. In general, levels of mildew showed an increase until early July (G.S. 40) then declined as chlorosis and necrosis increased. (Figure 9). Mildew levels at the first four assessments are, therefore, of greatest interest. Infection was generally very low on the youngest fully expanded leaf, especially at earlier times of assessment and was at its height as the flag leaf emerged. (Table 12). The patterns of mildew development for cultivars arrayed according to their different BMR Groups are shown in Figure 10.

Table 11. Spring barley cultivars assessed in Experiment 2c (1978).

BMR Group	Resistance gene	Cultivar name (and recognition letter).
0	-	Proctor (P), Golden Promise (G.P.)
2	Mlg	Zephyr (Z), Imber (I), Julia (J), Armelle (A), Berac (B)
4	Mlv	Vada (V), Mala Abed (MA), Lofa Abed (LA), Lami (Lm), Varunda (Vr)
5	Mlas	Sultan (S), Hassan (H)
6	Mla4/7	Ark Royal (AR), Wing (W), Tern (T)
2 + 4		Abacus (Ab), Luke (L), Universe (U), Sundance (Sn), Koru (K), Georgie (G)
3 + 4	Mla6	Yamina (Y)
2 + 5		Aramir (Ar), Athos (At), Porthos (Po)
2 + 5 + x		Maris Mink (MM)
2 + 6		Mazurka (Mz)
6 + x		Dram (D)

Figure 9. Average percentage mildew, chlorosis and necrosis for all cultivars on top four leaves (1-4) and mean (m) of all leaves.

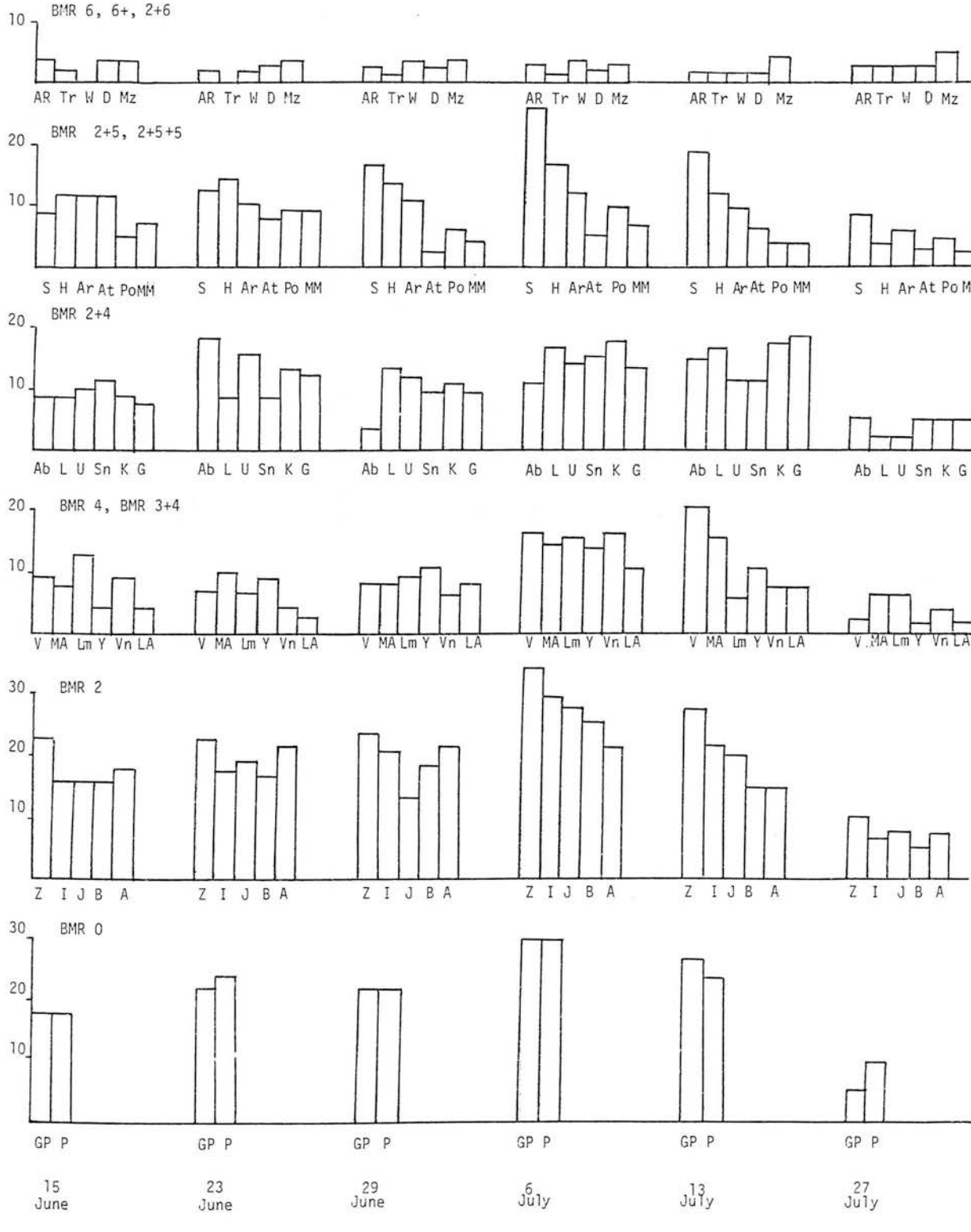


Higher disease levels were generally seen on cultivars in groups BMR0 and BMR2. Disease levels were intermediate for cultivars of BMR4 and 5 and very low in BMR6. High disease ratings were generally associated with a rapid development of chlorosis and necrosis.

Table 12. Average percentage of mildew (M), chlorosis (C) and necrosis (N) for all cultivars at each assessment date. (transformed values)

Date	Growth stage	Variate	Leaf Number 1 (top)	2	3	4	Mean
15 June	31	M	0	1	8	11	5
		C	0	0	1	1	1
		N	0	0	1	1	1
23 June	34	M	1	4	10	12	7
		C	0	1	3	7	3
		N	0	1	1	5	2
29 June	39	M	1	2	9	13	6
		C	1	1	2	4	2
		N	1	1	2	5	2
7 July	44	M	2	8	18	19	12
		C	1	2	4	8	3
		N	1	2	9	17	7
13 July	54	M	2	7	12	12	8
		C	1	4	9	13	6
		N	1	6	15	25	12
25 July	77	M	3	4	2	1	2
		C	2	10	16	15	10
		N	6	20	43	59	32

Figure 10. Percentage mildew (transformed values) for barley cultivars (mean of upper four leaves) - 1978.



BMRO.

Table 13 summarizes mildew development on Golden Promise and Proctor, both with no known major genes for resistance. No significant differences were found in mean values of the two cultivars over the assessment period. However, in looking at leaf position effects (Table 14) levels of mildew on the youngest leaf was less on Proctor than on Golden Promise. At the final assessment leaf necrosis levels of the earlier maturing cultivar, Golden Promise, were high, resulting in a low mildew score.

Table 13. Mean levels of mildew (M), chlorosis (C) and necrosis (N) on Golden Promise (G.P.) and Proctor (P). (D.F. = 145). Percentage (transformed values).

Date		Growth Stage	M	C	N
15 June	G.P.	31	15	2	0
	P	31	15	1	1
	S.E.D.±	1.6	2.2	1.0	1.1
23 June	G.P.	34	22	6	3
	P	32	23	5	4
	S.E.D.±	1.3	2.5	2.7	2.0
29 June	G.P.	40	22	13	3
	P	35	22	10	4
	S.E.D.±	2.5	2.7	1.9	2.2
6 July	G.P.	45	33	10	13
	P	40	33	12	19
	S.E.D.±	2.7	3.5	2.6	3.5
13 July	G.P.	56	27	14	31
	P	42	24	14	25
	S.E.D.±	2.5	3.2	3.5	3.7
27 July	G.P.	81	5	7	60
	P	68	9	9	51
	S.E.D.±	3.5	2.7	3.9	7.9

Table 14. Mean percentage (transformed values) of mildew chlorosis and necrosis on upper four leaves of Golden Promise (G.P.) and Proctor (P). (D.F. = 145).

Date		Mildew				Chlorosis				Necrosis			
		leaf position											
		1	2	3	4	1	2	3	4	1	2	3	4
15 June	G.P.	0	10	22	27	0	0	0	8	0	0	0	0
	P.	0	6	24	30	0	0	3	3	0	0	0	3
	S.E.D.±	0.0	3.3	4.1	4.3	0.0	0.0	1.6	3.3	0.0	0.0	0.0	3.1
23 June	G.P.	8	23	31	26	0	0	8	15	0	0	2	8
	P.	1	18	38	36	0	0	8	11	0	0	5	9
	S.E.D.±	1.4	4.3	4.8	5.4	0.0	0.0	4.8	6.6	0.0	0.0	3.7	5.6
29 June	G.P.	7	18	26	35	0	5	19	29	0	0	0	10
	P.	2	14	31	40	0	3	19	17	2	3	3	8
	S.E.D.±	1.7	2.9	4.8	5.2	0.0	2.1	3.3	4.2	0.6	1.5	3.8	5.8
6 July	G.P.	14	33	43	43	0	9	12	17	0	6	14	33
	P.	11	30	48	43	0	12	19	19	3	10	22	41
	S.E.D.±	3.5	5.2	5.3	5.9	1.0	3.5	5.3	6.0	1.3	3.9	6.3	7.5
13 July	G.P.	19	28	31	29	8	14	16	16	3	29	41	53
	P.	7	28	33	28	2	19	22	13	0	14	36	51
	S.E.D.±	3.3	5.3	5.1	5.2	2.3	4.9	6.1	7.1	2.5	5.2	5.7	8.1
27 July	G.P.	11	8	0	0	7	19	0	0	12	49	90	90
	P.	8	19	8	0	7	25	5	0	5	28	80	90
	S.E.D.±	4.4	4.8	4.5	2.6	3.8	7.0	7.6	8.6	7.6	10.1	12.1	13.0

BMR 2.

Virulence to overcome the resistance characteristics of this group was present resulting in high disease levels (Figure 10). There was variation within the group, however: Zephyr was always most severely infected (Table 15). Imber, Julia and Berac showed consistently less infection than Zephyr. Armelle gave similar results to Zephyr in early assessments, but, as plants approached ear emergence, the increase in mildew shown by other cultivars was not exhibited by Armelle which had the lowest infection levels at later growth stages. This late resistance was reflected in less rapid leaf senescence at grain development. Cultivars with a low average mildew infection over all four leaves showed low infection levels on all leaves but levels of infection were particularly low on younger leaves.

BMR 4 and 3+4.

Plants in Group BMR4 and Yamina (BMR 3+4) showed, overall, lower levels of infection than the previous two groups. (Figure 10, Table 16). The within group differences were less significant than previous groups although Lofa Abed had a lower level of infection, from early growth stages. The addition of resistance gene Mla6 in Yamina to Mlv associated with Group 4 plants, did not appear to confer any advantage to that cultivar

BMR 2+4.

The general levels of mildew on plants in this group did not vary significantly from those in Group 4 (Figure 10) and there were few within group differences (Table 17).

Table 15. Percentage of mildew, chlorosis and necrosis (transformed values) for varieties of BMR2 Group.

Date	Variety	Growth Stage	Mean of 4 leaves		
			Mildew	Chlorosis	Necrosis
15 June	Zephyr	31	18	2	1
	Imber	31	12	0	1
	Julia	31	12	1	0
	Berac	30	12	1	0
	Armelle	31	14	3	0
	S.E.D.±	1.6	2.2	1.1	1.1
23 June	Zephyr	35	21	4	0
	Imber	38	15	4	1
	Julia	35	16	7	3
	Berac	34	15	4	1
	Armelle	32	20	10	6
	S.E.D.±	1.3	2.5	2.7	2.0
29 June	Zephyr	35	25	4	5
	Imber	43	19	4	1
	Julia	41	13	3	2
	Berac	40	17	6	2
	Armelle	36	20	11	3
	S.E.D.±	2.5	2.7	1.9	2.2
6 July	Zephyr	45	35	12	14
	Imber	52	30	11	20
	Julia	42	29	11	17
	Berac	46	26	8	16
	Armelle	41	21	8	10
	S.E.D.±	2.7	3.5	2.6	3.5
13 July	Zephyr	56	28	14	24
	Imber	59	22	14	20
	Julia	55	21	18	21
	Berac	56	16	13	30
	Armelle	53	16	15	19
	S.E.D.±	2.2	3.2	3.5	3.7
27 July	Zephyr	81	12	6	60
	Imber	83	7	20	43
	Julia	81	8	18	39
	Berac	80	6	10	56
	Armelle	80	8	15	34
	S.E.D.±	3.5	2.7	3.9	7.9

(D.F. = 145)

Table 16. Percentage of mildew, chlorosis and necrosis (transformed values) for varieties of BMR4 and BMR3 + 4 groups.

Date	Variety	Growth stage	Mean of 4 leaves		
			Mildew	Chlorosis	Necrosis
15 June	Vada	30	9	1	1
	Mala Abed	28	7	0	1
	Lami	32	12	2	3
	Yamina	31	4	0	1
	Varunda	31	9	1	0
	Lofa Abed	31	4	1	1
	S.E.D.±	1.6	2.2	1.0	1.1
23 June	Vada	33	7	7	3
	Mala Abed	33	9	3	2
	Lami	34	7	9	4
	Yamina	33	8	3	2
	Varunda	34	4	4	3
	Lofa Abed	35	3	4	4
	S.E.D.±	1.3	2.5	2.7	2.0
29 June	Vada	42	6	1	2
	Mala Abed	38	6	0	2
	Lami	40	7	1	6
	Yamina	37	8	1	3
	Varunda	37	5	2	4
	Lofa Abed	37	0	0	4
	S.E.D.±	2.5	2.7	1.9	2.2
6 July	Vada	43	14	4	13
	Mala Abed	40	13	2	8
	Lami	44	14	8	13
	Yamina	41	12	6	11
	Varunda	40	14	5	11
	Lofa Abed	40	8	3	11
	S.E.D.±	2.7	3.5	2.6	3.5
13 July	Vada	55	18	15	16
	Mala Abed	48	13	6	12
	Lami	52	5	11	16
	Yamina	53	11	8	16
	Varunda	55	7	8	12
	Lofa Abed	48	7	6	14
	S.E.D.±	2.5	3.2	3.5	3.7
27 July	Vada	77	2	14	29
	Mala Abed	77	3	9	26
	Lami	75	3	17	27
	Yamina	74	1	20	25
	Varunda	71	2	14	34
	Lofa Abed	74	1	9	28
	S.E.D.±	3.5	2.7	3.9	7.9

(D.F. = 145)

Table 17. Percentage of mildew, chlorosis and necrosis (transformed values) for varieties of BMR 2+4 group.

Date	Variety	Growth stage	Mean of 4 leaves		
			Mildew	Chlorosis	Necrosis
15 June	Abacus	31	6	0	2
	Luke	29	6	0	1
	Universe	31	8	1	0
	Sundance	31	10	2	3
	Koru	31	7	0	1
	Georgie	31	6	0	1
	S.E.D.±	1.6	2.3	1.0	1.1
23 June	Abacus	33	15	5	7
	Luke	33	7	4	4
	Universe	32	12	9	8
	Sundance	35	7	2	3
	Koru	34	10	1	0
	Georgie	35	9	3	4
	S.E.D.±	1.3	2.5	2.7	2.0
29 June	Abacus	40	2	0	1
	Luke	38	9	0	3
	Universe	39	8	1	3
	Sundance	39	5	1	4
	Koru	38	7	0	2
	Georgie	40	5	0	2
	S.E.D.±	2.5	2.7	1.9	2.2
6 July	Abacus	44	8	5	6
	Luke	42	14	5	7
	Universe	41	11	5	10
	Sundance	43	13	5	11
	Koru	43	15	4	10
	Georgie	51	11	3	9
	S.E.D.±	2.7	3.5	2.6	3.5
13 July	Abacus	55	12	4	12
	Luke	52	13	13	15
	Universe	53	9	6	9
	Sundance	56	9	4	13
	Koru	52	15	7	18
	Georgie	56	17	8	13
	S.E.D.±	2.5	3.2	3.5	3.7
27 July	Abacus	82	4	9	25
	Luke	79	1	14	33
	Universe	75	1	16	24
	Sundance	79	4	15	36
	Koru	79	4	17	31
	Georgie	83	4	13	30
	S.E.D.±	3.5	2.7	3.9	7.9

(D.F. = 145)

BMR 5, 2+5. 2+5+.

Plants in this group showed a wider range of within-group variation than did the previous group (Figure 10, Table 18). Sultan and Hassan (Group 5) showed higher levels of mildew than Aramir, Athos or Porthos (Group 2+5) despite the apparent ineffectiveness of BMR2 resistance (Table 15). Maris Mink (Group 2+5+x) with additional resistance factors showed a consistently low level of infection. Comparing Sultan and Hassan, Sultan was infected to a greater degree than Hassan after stem elongation (G.S. 40) although both varieties matured at a similar rate.

BMR 6, 6+ and 2+6.

Mildew levels of cultivars in these groups were consistently low (Figure 10, Table 19) with no significant differences among cultivars.

Table 18. Percentage of mildew, chlorosis and necrosis (transformed values) for varieties of BMR5, 2+5 and 2+5+?

Date	Variety	Growth stage	Mean of 4 leaves		
			Mildew	Chlorosis	Necrosis
15 June	Sultan	31	9	2	1
	Hassan	31	10	0	2
	Aramir	31	10	1	2
	Athos	31	10	0	1
	Porthos	31	5	1	0
	Maris Mink	31	7	2	2
	S.E.D.±	1.6	2.2	1.0	1.1
23 June	Sultan	35	12	4	3
	Hassan	35	13	4	3
	Aramir	38	8	4	4
	Athos	34	6	7	5
	Porthos	33	8	4	4
	Maris Mink	32	8	3	3
	S.E.D.±	1.3	2.5	2.7	2.0
29 June	Sultan	40	14	4	3
	Hassan	40	11	5	6
	Aramir	40	9	5	3
	Athos	40	2	0	1
	Porthos	41	5	1	2
	Maris Mink	33	4	1	4
	S.E.D.±	2.5	2.7	1.9	2.2
6 July	Sultan	43	23	8	10
	Hassan	49	16	8	8
	Aramir	47	13	7	9
	Athos	46	7	3	10
	Porthos	46	11	6	9
	Maris Mink	41	8	4	10
	S.E.D.±	2.7	3.5	2.6	3.5
13 July	Sultan	58	20	14	18
	Hassan	57	13	16	17
	Aramir	55	11	11	17
	Athos	57	8	7	10
	Porthos	56	6	7	18
	Maris Mink	57	6	8	11
	S.E.D.±	2.5	3.2	3.5	3.7
27 July	Sultan	75	8	17	27
	Hassan	77	4	13	37
	Aramir	80	5	17	32
	Athos	82	2	14	31
	Porthos	80	4	14	41
	Maris Mink	82	1	16	29
	S.E.D.±	3.5	2.7	3.9	7.9

(D.F. = 145)

Table 19. Percentage of mildew, chlorosis and necrosis (transformed values) for varieties of BMR6, 6+ and 2+6 group.

Date	Variety	Growth stage	Mean of 4 leaves		
			Mildew	Chlorosis	Necrosis
15 June	Ark Royal	31	3	0	1
	Tern	31	1	0	2
	Wing	30	0	0	1
	Dram	31	2	0	0
	Mazurka	31	2	1	0
	S.E.D.±	1.6	2.2	1.0	1.0
23 June	Ark Royal	33	2	0	0
	Tern	34	0	1	1
	Wing	33	1	0	2
	Dram	33	2	1	1
	Mazurka	35	4	1	0
	S.E.D.±	1.3	2.5	2.7	2.0
29 June	Ark Royal	37	2	1	3
	Tern	40	1	0	1
	Wing	38	2	0	1
	Dram	38	1	1	2
	Mazurka	40	2	0	4
	S.E.D.±	2.5	2.7	1.9	2.2
6 July	Ark Royal	40	2	2	6
	Tern	45	1	0	3
	Wing	41	3	1	7
	Dram	41	1	2	5
	Mazurka	50	2	0	5
	S.E.D.±	2.7	3.5	2.6	3.5
13 July	Ark Royal	47	0	2	8
	Tern	62	1	1	7
	Wing	53	1	2	7
	Dram	56	1	3	6
	Mazurka	58	4	4	8
	S.E.D.±	2.5	3.2	3.5	3.7
27 July	Ark Royal	75	2	5	15
	Tern	83	2	9	23
	Wing	81	2	7	17
	Dram	81	1	11	15
	Mazurka	82	5	13	13
	S.E.D.±	3.5	2.7	3.9	7.9

(D.F. = 145)

Experiment 2d. Further assessments of mildew development on commercial cultivars of barley possessing different resistance genes.

Following the results of Experiment 2c, the variation of infection response to mildew over a wider range of cultivars was investigated.

Materials and methods.

In 1979, 48 barley cultivars were chosen and assigned to 12 groups listed in Table 20, according to their BMR categories (Wolfe and Slater, 1979). Plots consisted of cultivar groups arranged in a randomized block lay-out with six replicates. Individual cultivars formed sub-plots in a split-plot design. Each sub-plot consisted of about 10 plants grown in a 15 cm diameter circle, supported by a wire ring, with 15 cm spacing between clumps, as in Experiment 2c. Plants were sown on 5/6 April, 1979 and five assessments of mildew, chlorosis and necrosis were made from 16 May (G.S. 30) until 20 June (G.S. 60-70).

Results.

The results of mildew assessments in relation to leaf position and growth stage are given in Table 21. Little mildew appeared on the top leaves until after ear emergence when moderate increases were observed on the most susceptible cultivars. Differences in the results of the means of leaf positions for cultivar groups were generally reflected in the results for individual leaf positions, low infection rates for upper leaves being associated in particular with groups, E,F,J and K. No mildew was recorded on the upper leaves of plants in Group G. Results at different growth stages for all cultivars are summarized in Figure 11. The general pattern of development of mildew, chlorosis and necrosis was similar to that of the previous year.

Table 20. Spring barley cultivars assessed in Experiment 2d (1979).

Group	BMR Group	Resistance genes	Cultivar name (and recognition letter)
A	0	-	Golden Promise (G.P.) Proctor (P), Spratt Archer (S.A.), Ymer (Y)
B	2	Mlg	Armelle (A), Julia (J), Katy (Ka), Zephyr (Z)
C	2	-	Berac (B), Imber (I)
	3	Mla6	Maris Concord (MC), Midas (Mi)
D	4	Mlv	Lofa Abed (LA), Mala Abed (MA), Vada (V), Varunda (Vr)
E	5	Mla5	Baltsar (Ba), Hassan (H), Sultan (S),
	2+5	-	Piccolo (Pi)
F	6	Mla4/7	Firecrest (F), Keg (Ke), Tern (T), Wing (W)
G	8	Mla4/9	Akka (AK), Albion (Al), Simon (Si) Welam (We)
H	2+4	-	Abacus (Ab), Luke (L), Sundance (Sn), Universe (U)
I	2+5		Aramir (Ar), Athos (At), Porthos (Po),
	2+5+		Maris Mink (MM)
J	6		Ark Royal (AR)
	6+		Dram (D), Triumph (Tr)
	2+6		Mazurka (Mz)
K	3+4		Goldspear (Gd), Jupiter (Ju), Minak (Mk), Yamina (Y)
L	4		Ambre (AM, Lami (Lm)
	2+4		Georgie (G)
	3+4		Goldmarker (Gm)

Table 21. Percentage mildew (transformed values) for cultivar groups in relation to leaf position and growth stage.

Leaf Position	GROUP A	B	C	D	E	F	G	H	I	J	K	L	S.E.D.± (DF=55)
GS 30													
1(top)	1	0	0	0	0	0	1	1	0	0	0	0	0.4
2	3	1	3	1	0	1	0	3	0	0	1	1	1.3
3	12	11	11	10	10	9	1	9	10	9	7	8	3.2
4	20	19	16	16	15	12	3	14	16	15	14	12	3.7
Mean	8	8	7	7	6	5	1	7	6	6	6	5	1.8
GS 38													
1	0	0	1	0	0	0	0	0	0	0	0	0	0.2
2	6	8	9	11	5	4	0	8	6	5	6	8	2.7
3	28	27	29	27	21	19	0	23	18	21	22	21	3.5
4	36	34	37	32	28	27	5	31	26	25	32	28	3.5
Mean	18	17	19	17	13	12	1	16	25	13	15	14	2.1
GS 48													
1	11	3	4	0	3	0	0	2	1	1	1	1	1.6
2	10	13	9	5	3	1	0	10	6	3	1	6	2.5
3	20	25	21	16	15	11	1	22	13	14	10	19	2.7
4	25	24	21	23	24	9	2	23	18	21	21	26	3.4
Mean	14	16	14	11	11	8	1	14	10	8	8	13	1.7
GS 56													
1	6	4	8	2	3	1	0	6	2	2	0	1	2.1
2	17	17	16	11	11	7	0	15	8	6	10	10	3.1
3	23	24	19	18	17	13	1	20	15	17	15	16	3.0
4	17	18	10	16	14	11	5	9	12	15	15	12	3.5
Mean	21	21	26	19	22	18	4	27	22	20	20	21	3.0
GS 67													
1	7	9	8	3	7	3	0	7	3	2	4	8	2.2
2	17	21	13	10	13	6	0	13	7	8	8	16	3.1
3	15	13	5	5	11	6	0	8	8	8	8	6	2.9
4	7	4	2	1	2	3	0	3	5	2	4	1	1.9
Mean	11	17	7	5	8	6	0	8	6	5	6	8	1.7

Figure 11. Percentage mildew (transformed values) for barley cultivars (mean of upper four leaves) - 1979.

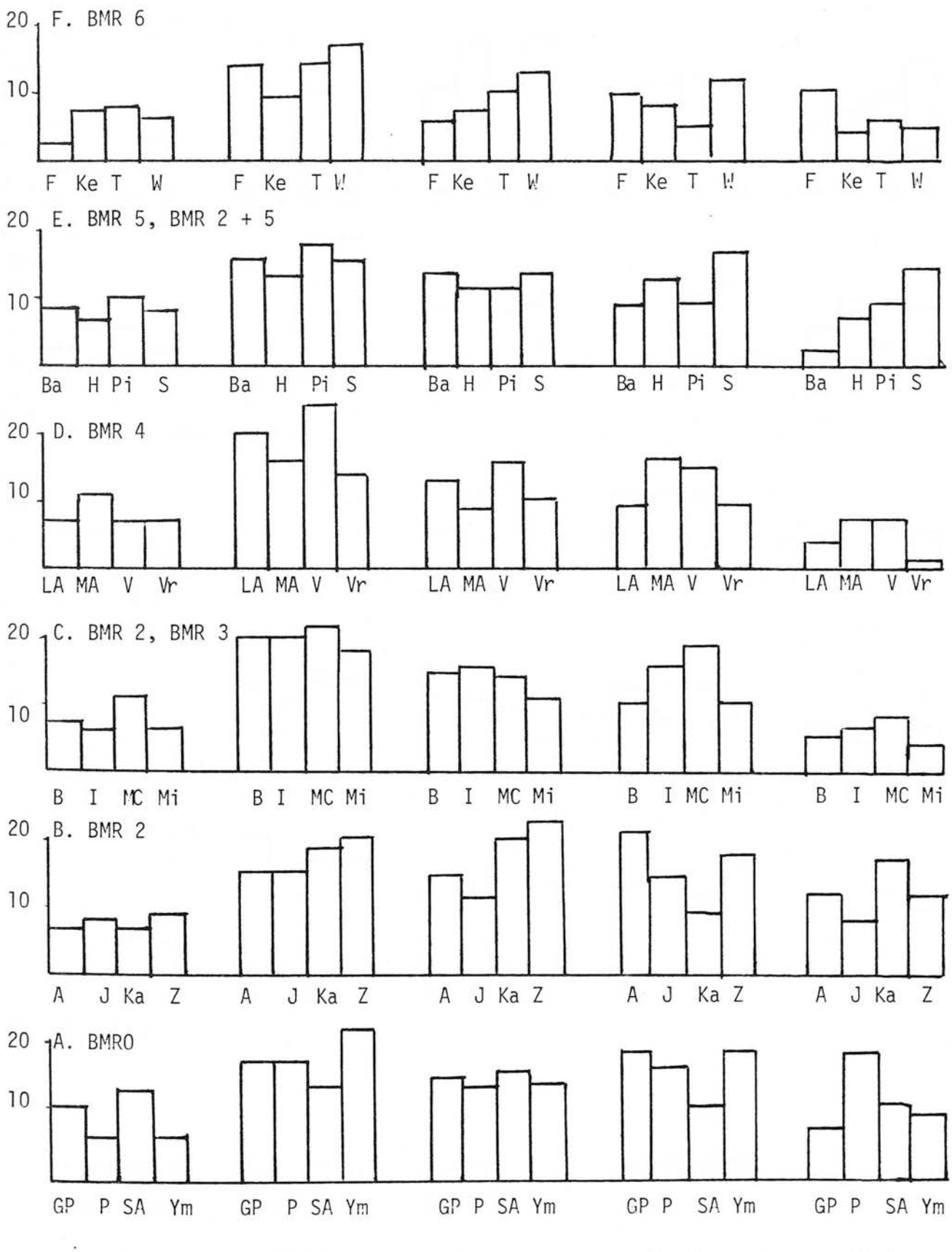
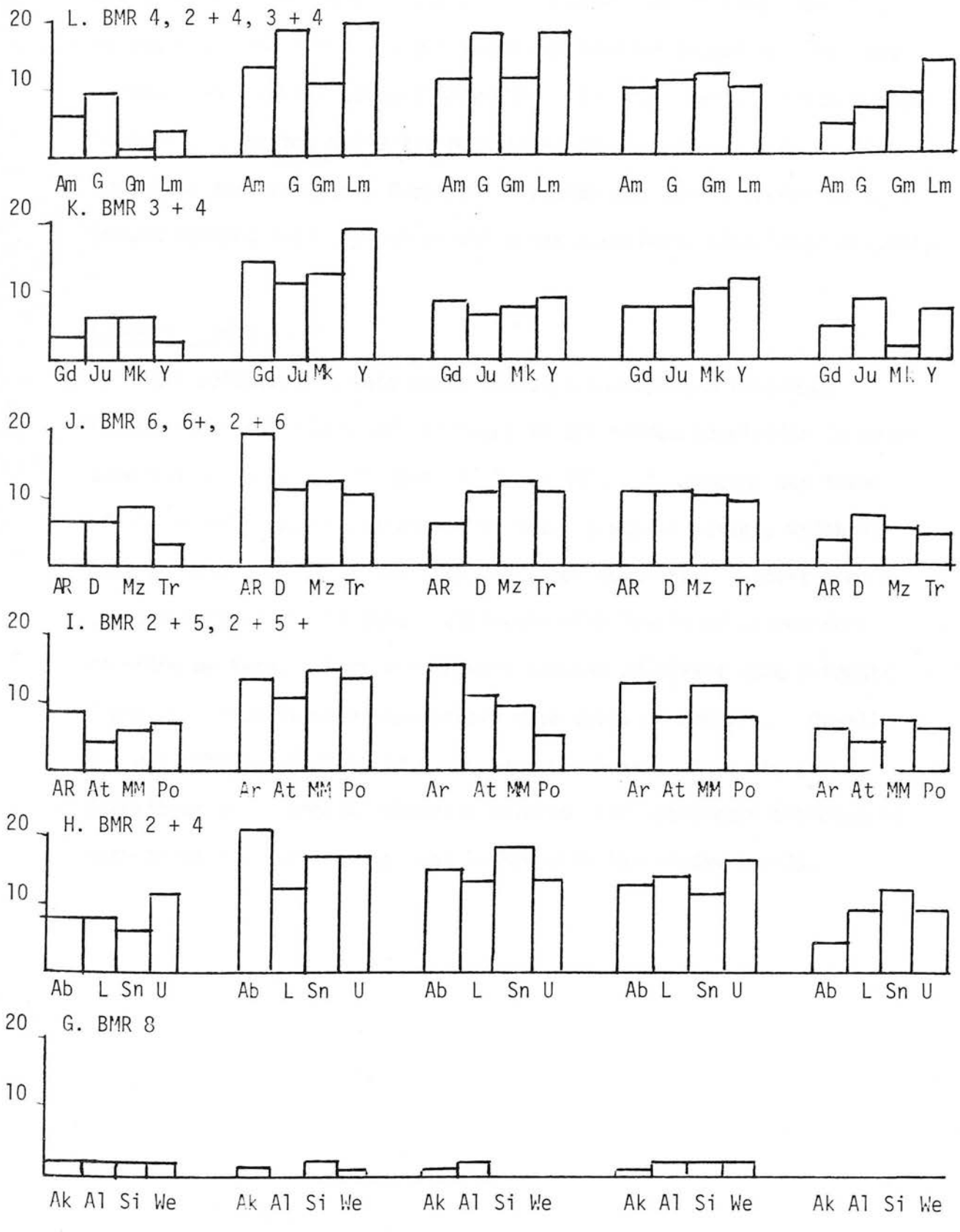


Figure 11. Percentage mildew (transformed values) for barley cultivars (mean of upper four leaves) - 1979.



Group A. BMR0.

No cultivar in this group had any known major gene resistances. Mildew levels averaged for the upper four leaves showed no consistent variation with cultivar (Table 22). However, as in Experiment 2c, it was observed that the upper leaves of Proctor tended to show less mildew than Golden Promise (Table 23). In this respect Spratt Archer behaved as Proctor, while the pattern of disease development of Ymer resembled that of Golden Promise. Proctor and Spratt Archer both showed delayed leaf senescence which was associated with later maturity.

Group B. BMR2.

All cultivars in this group showed a susceptible response, indicating the presence of virulence in the mildew population to overcome Mlg resistance (Figure 11, Table 24). No account was taken of the vernalisation requirements of Katy, a winter barley, which in consequence, did not mature with the other cultivars. Apart from an anomalous result on 13 June, relatively high levels of mildew were recorded on Katy. Some differences between cultivars were evident; Zephyr was usually more susceptible than Julia or Armelle. Armelle did not exhibit adult plant resistance as clearly as it had in Experiment 2c. Armelle showed a delayed leaf senescence although it matured early, but this was not linked with low mildew levels.

Table 22. Percentage mildew (M), chlorosis (C), and necrosis (N) (transformed values) for group A cultivars in relation to leaf position (D.F. = 180).

Leaf Position	Golden Promise			Proctor			Spratt Archer			Ymer		
	M	C	N	M	C	N	M	C	N	M	C	N
16 May												
1 (top)	2	0	0	0	0	0	1	0	0	0	0	0
2	6	0	0	0	0	0	6	0	0	0	0	0
3	12	0	6	1	3	9	18	3	3	10	2	6
4	22	6	9	18	3	9	26	3	8	15	5	14
S.E.D.±	2.8	0.8	2.3	2.8	0.8	2.3	2.8	0.8	2.3	2.8	0.8	2.3
30 May												
1	0	0	0	0	0	0	0	0	0	0	0	0
2	5	0	0	0	0	0	2	0	0	13	0	0
3	28	3	5	25	17	9	25	8	3	33	22	12
4	34	19	6	42	15	15	27	21	8	43	22	23
S.E.D.±	3.3	2.2	2.5	3.3	2.2	2.5	3.3	2.2	2.5	3.3	2.2	2.5
6 June												
1	1	0	0	1	0	0	0	0	0	0	0	0
2	11	0	5	7	3	3	8	0	0	8	6	3
3	19	17	17	17	6	8	22	16	16	22	6	3
4	23	22	25	26	17	17	29	18	16	29	26	24
S.E.D.±	2.9	2.4	5.6	2.9	2.4	5.6	2.9	2.4	5.6	2.9	2.4	5.6
13 June												
1	11	3	8	0	2	0	2	0	0	9	0	0
2	17	15	17	14	5	11	13	16	11	21	7	3
3	28	24	28	21	19	20	21	22	26	21	23	16
4	16	13	63	23	24	31	8	6	60	21	16	40
S.E.D. ±	5.9	2.6	5.7	5.9	2.6	5.7	5.9	2.6	5.7	5.9	2.6	5.7
20 June												
1	10	4	5	3	0	3	0	3	0	15	13	14
2	10	26	28	20	12	19	19	16	11	18	23	38
3	8	18	50	25	28	33	19	24	33	8	18	63
4	0	3	85	22	19	55	6	16	51	8	0	90
S.E.D.±	3.0	2.6	7.2	3.0	2.6	7.2	3.0	2.6	7.2	3.0	2.6	7.2

Table 23. Percentage of mildew, chlorosis and necrosis (transformed values) for Group A cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves		
			Mildew	Chlorosis	Necrosis
16 May	Golden Promise	30	10	2	4
	Proctor	30	6	2	5
	Spratt Archer	30	13	2	3
	Ymer	30	6	2	5
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Golden Promise	38	17	6	3
	Proctor	37	17	8	6
	Spratt Archer	38	13	7	3
	Ymer	37	22	11	9
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Golden Promise	46	14	10	12
	Proctor	43	13	7	7
	Spratt Archer	40	15	9	8
	Ymer	44	14	9	11
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Golden Promise	60	18	14	29
	Proctor	49	15	13	16
	Spratt Archer	48	11	11	24
	Ymer	54	18	12	15
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Golden Promise	65	7	13	42
	Proctor	55	18	15	28
	Spratt Archer	53	11	15	24
	Ymer	75	10	14	51
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Table 24. Percentage of mildew, chlorosis and necrosis (transformed values) for Group B cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Armelle	30	7	0	4
	Julia	30	8	1	3
	Katy	30	7	2	5
	Zephyr	30	9	1	2
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Armelle	34	15	5	5
	Julia	39	15	7	5
	Katy	31	18	6	3
	Zephyr	41	20	3	1
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Armelle	45	14	7	10
	Julia	45	11	10	12
	Katy	34	19	10	7
	Zephyr	53	22	9	11
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Armelle	53	20	17	18
	Julia	57	14	13	27
	Katy	36	9	9	19
	Zephyr	60	18	12	20
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Armelle	75	12	15	27
	Julia	67	8	12	46
	Katy	34	16	12	25
	Zephyr	70	11	8	46
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Group C. BMR2, BMR3.

There were no consistent differences between group C cultivars (Figure 11, Table 25). Only at the time of inflorescence emergence did levels vary slightly, Berac (Mlg) and Midas (Mla6) showing less infection than Imber (Mlg) and Maris Concord (Mla6).

Table 25. Percentage of mildew, chlorosis and necrosis (transformed values) for Group C cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Berac	30	7	1	5
	Imber	31	6	1	4
	Maris Concord	30	11	2	5
	Midas	30	6	0	0
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Berac	36	19	7	7
	Imber	44	19	3	2
	Maris Concord	38	20	4	5
	Midas	37	17	4	4
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Berac	49	14	9	10
	Imber	54	15	11	8
	Maris Concord	45	14	8	27
	Midas	46	11	5	10
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Berac	59	10	12	20
	Imber	55	15	11	25
	Maris Concord	55	18	11	33
	Midas	54	10	12	26
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Berac	65	6	9	46
	Imber	75	7	10	45
	Maris Concord	68	9	9	55
	Midas	61	5	12	40
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Group D. BMR4.

All cultivars in this group became substantially infected with mildew (Figure 11, Table 26). Differences within the group were small although Vada tended to show more infection, Varunda less.

Table 26. Percentage of mildew, chlorosis and necrosis (transformed values) for Group D cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Lofa Abed	30	6	0	2
	Mala Abed	30	9	1	2
	Vada	30	6	0	3
	Varunda	31	6	1	5
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Lofa Abed	38	19	7	5
	Mala Abed	37	15	5	4
	Vada	37	23	6	6
	Varunda	44	13	7	6
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Lofa Abed	48	12	9	8
	Mala Abed	47	8	4	4
	Vada	47	14	6	6
	Varunda	51	9	6	12
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Lofa Abed	52	8	12	20
	Mala Abed	56	15	15	21
	Vada	54	14	10	21
	Varunda	57	9	10	16
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Lofa Abed	63	4	9	38
	Mala Abed	62	7	9	35
	Vada	68	7	11	41
	Varunda	65	1	12	38
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Group E. BMR5, BMR 2+5.

Baltsar, Hassan and Sultan represented cultivars with resistance gene Mlas while Piccolo contained resistance Mlas and Mlg. Piccolo was later maturing than the other cultivars and showed less necrosis at the final assessment, although levels of mildew were broadly similar to those on the other cultivars. Sultan had a tendency to develop more mildew than other cultivars at the later growth stages (Figure 11, Table 27).

Table 27. Percentage of mildew, chlorosis and necrosis (transformed values) for Group E cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Baltsar	30	6	0	3
	Hassan	30	5	1	4
	Piccolo	30	8	1	4
	Sultan	30	6	0	2
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Baltsar	40	13	5	6
	Hassan	39	11	3	3
	Piccolo	35	16	8	3
	Sultan	40	14	3	4
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Baltsar	51	12	6	13
	Hassan	48	10	8	7
	Piccolo	44	10	6	14
	Sultan	48	12	7	7
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Baltsar	64	8	9	27
	Hassan	60	11	11	16
	Piccolo	53	8	11	24
	Sultan	58	16	12	19
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Baltsar	77	3	11	40
	Hassan	75	7	14	38
	Piccolo	68	9	14	26
	Sultan	69	14	14	37
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Group F. BMR6.

All cultivars in this group were moderately affected with mildew (Figure 11, Table 28) and there were no consistent differences, apart from slightly higher levels of mildew being recorded on Wing from booting (G.S. 40) to flowering (G.S. 60) stages.

Table 28. Percentage of mildew, chlorosis and necrosis (transformed values) for Group F cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Firecrest	30	2	0	2
	Keg	30	6	0	2
	Tern	30	8	0	2
	Wing	31	6	0	2
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Firecrest	40	12	8	10
	Keg	41	8	4	4
	Tern	38	13	5	3
	Wing	37	16	7	4
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Firecrest	45	5	3	20
	Keg	45	6	7	8
	Tern	47	9	7	7
	Wing	45	11	6	5
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Firecrest	57	8	6	24
	Keg	61	7	7	15
	Tern	64	5	9	15
	Wing	59	11	11	16
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Firecrest	55	7	14	24
	Keg	69	3	11	24
	Tern	74	5	11	32
	Wing	71	4	9	39
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Group G. BMR8.

Mildew levels in this group of cultivars possessing Mla4/9 were too low to detect variation in background resistance (Figure 11, Table 29).

Table 29. Percentage of mildew, chlorosis and necrosis (transformed values) for Group C cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Akka	31	1	0	0
	Albion	31	1	0	1
	Simon	30	1	0	2
	Welam	30	1	0	2
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Akka	49	1	0	0
	Albion	42	0	1	1
	Simon	39	3	1	1
	Welam	39	1	0	1
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Akka	56	1	1	1
	Albion	51	2	2	5
	Simon	47	0	0	2
	Welam	48	0	0	1
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Akka	60	1	2	3
	Albion	58	2	2	4
	Simon	58	2	1	6
	Welam	57	2	2	2
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Akka	82	0	7	3
	Albion	67	0	4	3
	Simon	76	0	2	2
	Welam	68	0	1	3
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Group H. BMR 2+4.

Cultivars in this group with combined resistance factors Mlg and Mlv showed some variation (Figure 11, Table 30). Luke and Sundance tended to be slightly less infected during active growth stages. Cultivars in this group, however, did not have any obvious advantage over cultivars with either resistance factor alone e.g. Armelle, Mlg (Table 24), or Vada, Mlv (Table 26).

Table 30. Percentage of mildew, chlorosis and necrosis (transformed values) for Group H cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Abacus	30	6	0	4
	Luke	30	6	0	4
	Sundance	30	5	0	3
	Universe	30	10	2	0
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Abacus	40	19	5	4
	Luke	38	11	4	5
	Sundance	39	16	5	3
	Universe	37	16	7	5
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Abacus	52	14	10	8
	Luke	47	12	5	5
	Sundance	49	17	9	6
	Universe	43	14	8	6
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Abacus	59	12	14	28
	Luke	60	13	10	31
	Sundance	60	10	8	20
	Universe	56	15	11	29
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Abacus	76	4	11	37
	Luke	69	8	11	34
	Sundance	66	11	14	43
	Universe	68	8	10	30
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Group I. BMR 2+5, BMR 2+5+.

Cultivars with Mlg + Mlas resistance (Figure 11, Table 31) showed mildew levels similar to group E with Mlas alone (Table 27). Aramir did show slightly more infection at inflorescence emergence (G.S. 50) than other cultivars in the group.

Table 31. Percentage of mildew, chlorosis and necrosis (transformed values) for Group I cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Aramir	30	8	1	2
	Athos	31	4	1	2
	Maris Mink	30	6	1	3
	Porthos	30	7	0	1
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Aramir	39	13	9	6
	Athos	40	10	3	4
	Maris Mink	36	14	8	6
	Porthos	39	13	2	4
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Aramir	52	15	10	11
	Athos	51	10	5	10
	Maris Mink	47	9	6	7
	Porthos	51	5	5	9
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Aramir	60	12	10	24
	Athos	58	7	6	22
	Maris Mink	56	12	15	22
	Porthos	59	7	9	23
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Aramir	69	6	11	42
	Athos	67	4	10	31
	Maris Mink	67	7	14	28
	Porthos	73	6	9	44
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Group J. BMR6, BMR 6+, BMR 2+6.

Mildew levels on plants in this group (Figure 11, Table 32) were similar to those of cultivars in group F (BMR6, Table 28) despite the presence of Ml_a resistance additional to Mla4/7 in Dram, Mazurka and Triumph: these were slightly less infected at early growth than Ark Royal but these effects were not sustained.

Table 32. Percentage of mildew, chlorosis and necrosis (transformed values) for Group J cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Ark Royal	30	6	0	1
	Dram	31	6	0	1
	Mazurka	30	8	2	4
	Triumph	30	3	0	2
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Ark Royal	35	18	9	8
	Dram	37	11	3	5
	Mazurka	42	12	6	9
	Triumph	39	10	3	6
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Ark Royal	47	6	4	7
	Dram	47	10	8	7
	Mazurka	51	12	8	8
	Triumph	51	10	5	11
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Ark Royal	50	11	12	16
	Dram	58	11	13	21
	Mazurka	57	10	13	23
	Triumph	66	9	8	20
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Ark Royal	59	4	8	41
	Dram	68	7	12	36
	Mazurka	77	6	12	37
	Triumph	72	5	9	39
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Group K. BMR 3+4.

Cultivars in this group with resistance factors Mla6 + Mlv showed slightly less infection (Figure 11, Table 33) than cultivars with either resistance factor alone (Tables 25 and 26). At stem elongation (G.S. 40) Jupiter and Minak both had relatively low levels of mildew, but this trend was not consistent at all assessments.

Table 33. Percentage of mildew, chlorosis and necrosis (transformed values) for Group K cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Goldspear	31	4	0	1
	Jupiter	30	7	0	4
	Minak	31	7	0	1
	Yamina	30	4	0	2
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Goldspear	38	15	3	4
	Jupiter	36	12	4	4
	Minak	37	13	4	2
	Yamina	37	20	3	1
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Goldspear	45	9	7	6
	Jupiter	44	7	5	6
	Minak	48	8	8	13
	Yamina	47	9	6	6
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Goldspear	52	8	10	21
	Jupiter	54	8	10	21
	Minak	53	11	12	17
	Yamina	52	12	11	24
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Goldspear	60	5	15	29
	Jupiter	70	9	11	33
	Minak	62	2	9	37
	Yamina	76	8	11	36
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

Group L. BMR4, BMR 2+4, BMR 3+4.

Levels of infection were moderate (Figure 11, Table 34) in this group. Goldmarker (M1v + M1a6) tended to show less infection than other cultivars until inflorescence emergence (G.S. 50); Ambre was less affected during stem elongation and booting.

Table 34. Percentage of mildew, chlorosis and necrosis (transformed values) for Group L cultivars.

Date	Cultivar	Growth stage	Mean of 4 leaves Mildew	Chlorosis	Necrosis
16 May	Ambre	30	6	1	5
	Georgie	30	8	0	1
	Goldmarker	30	1	0	3
	Lami	30	4	2	4
	S.E.D.±	0.2	1.8	0.5	1.4
30 May	Ambre	37	12	4	3
	Georgie	42	17	5	4
	Goldmarker	37	10	2	1
	Lami	38	18	7	8
	S.E.D.±	1.1	2.1	1.4	1.4
6 June	Ambre	48	10	7	7
	Georgie	54	16	5	7
	Goldmarker	48	10	2	2
	Lami	49	17	13	11
	S.E.D.±	1.5	1.7	1.1	3.0
13 June	Ambre	57	9	10	19
	Georgie	60	10	10	23
	Goldmarker	58	11	10	19
	Lami	54	9	11	24
	S.E.D.±	1.8	3.0	1.4	3.0
20 June	Ambre	67	4	10	41
	Georgie	67	6	7	46
	Goldmarker	72	8	9	34
	Lami	64	13	12	48
	S.E.D.±	3.4	1.8	1.4	4.0

(D.F. = 180)

DISCUSSION.

While the first series of investigations were concerned primarily with examining non-commercial barley lines for sources of mildew resistance, this second series considered the characteristics of cultivars which either had been or are still in commercial use. Again, interest was centred on intermediate forms of resistance. From a study of mainly older, out-dated cultivars, which had no known major genes for resistance, considerable variation in level of susceptibility was found. Asse showed remarkably low levels of infection during the investigation and indeed has been shown to have good field resistance (Russell, 1978). In this study Asse was grown in the glasshouse alongside cultivars from BMR groups 1-7 which all showed susceptible responses indicating a wide range of virulences in the pathogen population. A parallel trial was carried out on cultivars with Mlg resistance which was the first major gene to be extensively used in commercial situations and which has since been rendered ineffective by the emergence of virulent genotypes in the pathogen population. Cultivars with Mla6 resistance were also tested in this trial, this form of resistance again having been eroded following the introduction of Midas on a large scale. The generally high levels of infection for these cultivars pointed to the widespread occurrence in natural populations of the pathogen virulences to overcome the resistance factors of BMR 2 and 3 groups. It is of interest to note that often the disease levels were higher for these cultivars than for those from the previous group with no known major genes for resistance; this may relate to the masking of background resistances in the development of cultivars with major gene resistance. Variations in the performance of cultivars with BMR 2 or 3 resistance factors were observed.

Gerkra and Goldfoil proved to be generally more susceptible and Armelle more resistant than most cultivars. Howard, Johnson, Russell and Wolfe (1970) reported that Deba Abed was moderately resistant while Zephyr was highly susceptible but in the present study both cultivars behaved similarly.

In the third series of studies, commercial cultivars from several different BMR groups were assessed over two years. From the results, responses could be broadly termed highly susceptible, intermediately resistant and highly resistant. Groups of cultivars with the same BMR factors did not necessarily behave consistently over the two years and these group responses may be attributed to major gene effects (Figures 10 and 11). Thus the low level of disease in Ark Royal, Tern, Wing, Dram and Mazurka in the first year is associated with Mla4/7 resistance and the presumed low frequency of the relevant virulence factors in the pathogen population to overcome the resistance. The same cultivars were more substantially infected in the following year, indicating an increase in group 6 virulences. In the second year, cultivars with resistance genes Mla4/9, which had not been assessed in the previous year, showed only traces of infection: this may again be attributed to a major gene effect in the absence of the complementary genes for virulence in the pathogen population.

The intermediate levels of resistance recorded for cultivars with major gene resistance factors probably reflect the relative frequencies of complementary virulences in the local mildew population. Thus, in the first year, 1978, virulences to overcome BMR groups 4 (Mlv), 3 + 4 (Mla6 + Mlv), 2 + 4 (Mlg + Mlv), 5 (Mlas) or 2 + 5 (Mlg + Mlas) were less frequent than virulence for BMR 2 (Mlg). Cultivars belonging to BMR 2 showed levels of disease comparable to those of cultivars with no known major gene resistance; the virulence corresponding to Mlg would

therefore appear ubiquitous. In the second year, 1979, virulence genotypes to overcome Mla4/7, Mlg + Mlas or Mla6 + Mlv seemed to be less frequent than those for Mla6, Mlv, Mlas or Mlg + Mlv. Again, no advantage was conferred by Mlg resistance alone. The progressive increase in Mlg virulence with Mlg cultivars has been described by Wolfe and Schwarzbach (1978).

Within BMR groups some variation was observed in disease levels among cultivars, which may be attributed to background resistance. This was not consistent between years or within growing seasons. Such factors as the nutritional status of the host, light and plant age (Gaumann, 1950) may modify levels of intermediate resistance while Wolfe and Schwarzbach (1978) have indicated specificity among pathogen isolates to particular cultivars with different "background" genes. Howard, Johnson, Russell and Wolfe (1970) illustrated this with the cultivar Proctor, which was shown to decline in its resistance rating (N.I.A.B. recommended lists) over the period 1953 to 1969. It is, however, difficult to relate changes in resistance not conditioned by major genes to variations in the pathogen population. In this study the following trends were observed within groups.

In the BMRO group of cultivars both Proctor and Golden Promise showed high general levels of infection, although in comparing these cultivars, the onset of mildew colony development on the younger leaves appeared to be delayed with Proctor. Little and Doodson (1972) reported that Proctor did not show the same yield reduction relative to mildew infection that other cultivars did and delayed mildew development may be a factor explaining this. In the group BMR 2 less infection occurred on Armelle than Zephyr in both years. In some instances this seemed to be associated with adult plant resistance and may confer a yield advantage. Henness, (1978 unpublished) showed

that Armelle out-yielded Zephyr. Mildew on Armelle remained at 12% while progressing from 13% (G.S. 30) to 26% (G.S. 38) on Zephyr. There was no significant difference between the yields of the two cultivars when a fungicide treatment was applied. With cultivars in the BMR 4 and BMR 5 groups, slight differences were observed: Vada appeared more susceptible than Lofa Abed and Varunda, and Hassan was less infected than Sultan at later growth stages.

EXPERIMENTAL STUDIES 3.

Monocyclic tests on the development of *Erysiphe graminis* on leaves of different barley cultivars.

EXPERIMENTAL STUDIES 3.

Monocyclic tests on the development of *Erysiphe graminis* on leaves of different barley cultivars.

Experiments in the previous section have demonstrated the variation in response of commercial cultivars with known major gene resistance in the presence of a pathogen population with complex or mixed virulences. From a study on primary infection of wheat and barley by *E. graminis*, Masri and Ellingboe (1966) indicated that all major genes for resistance exhibited more than one effect in their mode of action - they may cause:

a) exclusion of the pathogen from the host cell;

b) delay in early haustorial development;

c) distortion of some haustoria;

d) destruction and distortion of the majority of haustoria 5 days after inoculation;

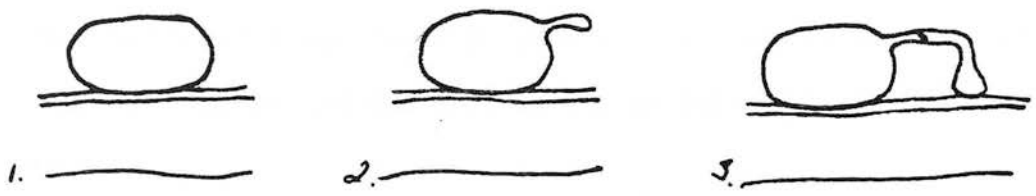
e) suppression of fungus sporulation after the establishment of primary infection;

thereby preventing the occurrence of repeating disease cycles.

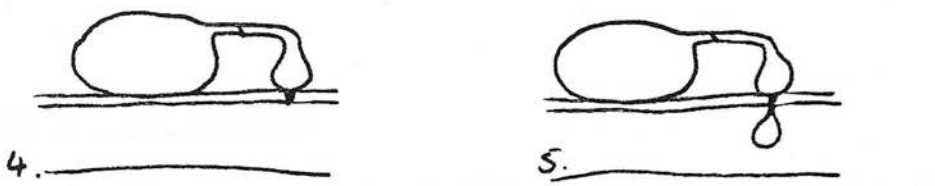
Partial resistance may also be said to be due to a number of components, affecting the infection ratio, sporulation rate, latent period or infectious period. The infection ratio is the fraction of spores that produce sporulating lesions in a disease-free crop, while the sporulation rate may be defined as the number of spores produced per lesion per day. Increased resistance may be associated with a decreased infection ratio, an increased latent period, a decreased infectious period or a combination of these factors (Zadoks, 1971).

Ellingboe (1972) characterised the distinct morphological stages of mildew establishment (Figure 12): the stage at which a

Figure 12. Morphological stages of primary infection by Erysiphe graminis (after Ellingboe, 1972).

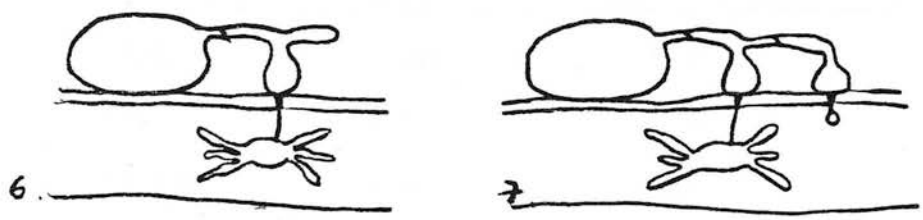


ungerminated spore spore germination formation of appressorium



formation of penetration peg piercing host cuticle and epidermal cell wall

formation of haustorium



establishment of functional haustorium and formation of secondary hyphal initials

formation of secondary hyphae capable of initiating secondary appressoria and haustoria.

particular resistance mechanism may act can be determined when the development of a fungal spore from a controlled inoculation is followed in a monocyclic test.

The aims of the experiments in this section were to look in detail at the fungal development on different barley cultivars after inoculation with spores of *E. graminis*, at the factors conditioning this development and how they relate to intermediate forms of resistance.

Experiment 3a. The development of known physiologic races of *Erysiphe graminis* on commercial cultivars of spring barley.

In this investigation the responses of *E. graminis* with different virulence factors on cultivars with different resistance factors were observed microscopically.

Materials and methods.

Five spring barley cultivars; Golden Promise, Wing, Hassan, Midas and Maris Mink, were inoculated with four races of *E. graminis*, provided by P.B.I., Cambridge, each known to be specifically virulent against one or other of the cultivars (see below):

major gene resistance	cultivar	race with equivalent virulence
Mla6	Midas	MC6
Mla6	Midas	XC8
Mla4/7	Wing	WC1
Mlas	Hassan	HC1

An isolate of unknown virulence combination from Golden Promise, designated race G.P., was also used. All races were maintained on excised leaf segments of their compatible host and spores not more

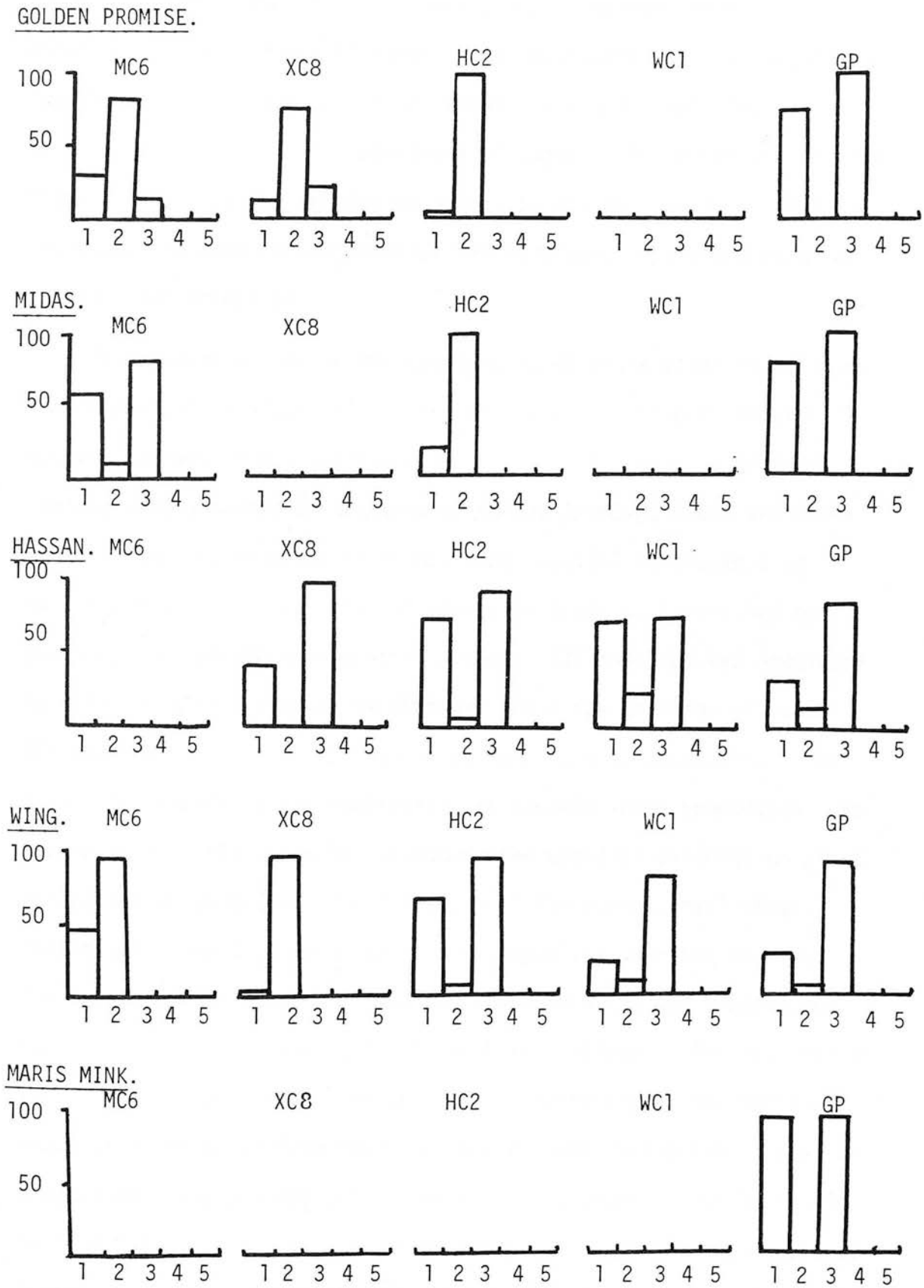
than 24 hours old were used in inoculations using a camel hair brush.

Plants were grown in an isolation propagator until G.S. 31 when standard 3 cm segments were cut from the uppermost fully expanded leaf. Samples of each cultivar, Midas, Wing, Hassan, Golden Promise (susceptible comparison) and Maris Mink (resistant comparison) were allocated to plastic dishes on benzimidazole agar and inoculated with each isolate of *E. graminis*. The plates were randomly assigned to an incubator maintained at a temperature of 18°C and illuminated with daylight category by fluorescent lamp strips for 16 hours in each 24 hour cycle. After 12 hours, 48 hours and 108 hours, three replicates of each combination were removed and the segments prepared for microscopic assessment by clearing in Carnoy's solution and staining with lactophenol cotton blue. A minimum of 100 spores were assessed.

Results.

Observations made on fungal development after 12 hours are summarized in Figure 13. The isolate G.P. from Golden Promise usually showed the highest rate of germination of any race on any cultivar: only this isolate was observed to have germinated on Maris Mink at this assessment. In most cases where germination had occurred to any extent, germ tubes had developed appressoria within the 12 hour period. Race MC6 germinated moderately well on Golden Promise, Midas and Wing but no appressoria were observed on Wing and only a few were seen on Golden Promise. On Midas, the compatible host of Race MC6, most germ tubes had formed appressoria. Race XC8, also isolated from Midas, had not developed on this host or on Maris Mink: of the other three cultivars, germination was poor on Golden Promise and Wing and the greatest development was observed on Hassan. Race HC2 had a high rate of germination on Hassan, its compatible host, and also on Wing:

Figure 13. Development of Erysiphe graminis on leaf segments 12 hours after inoculation in relation to cultivar and isolate.

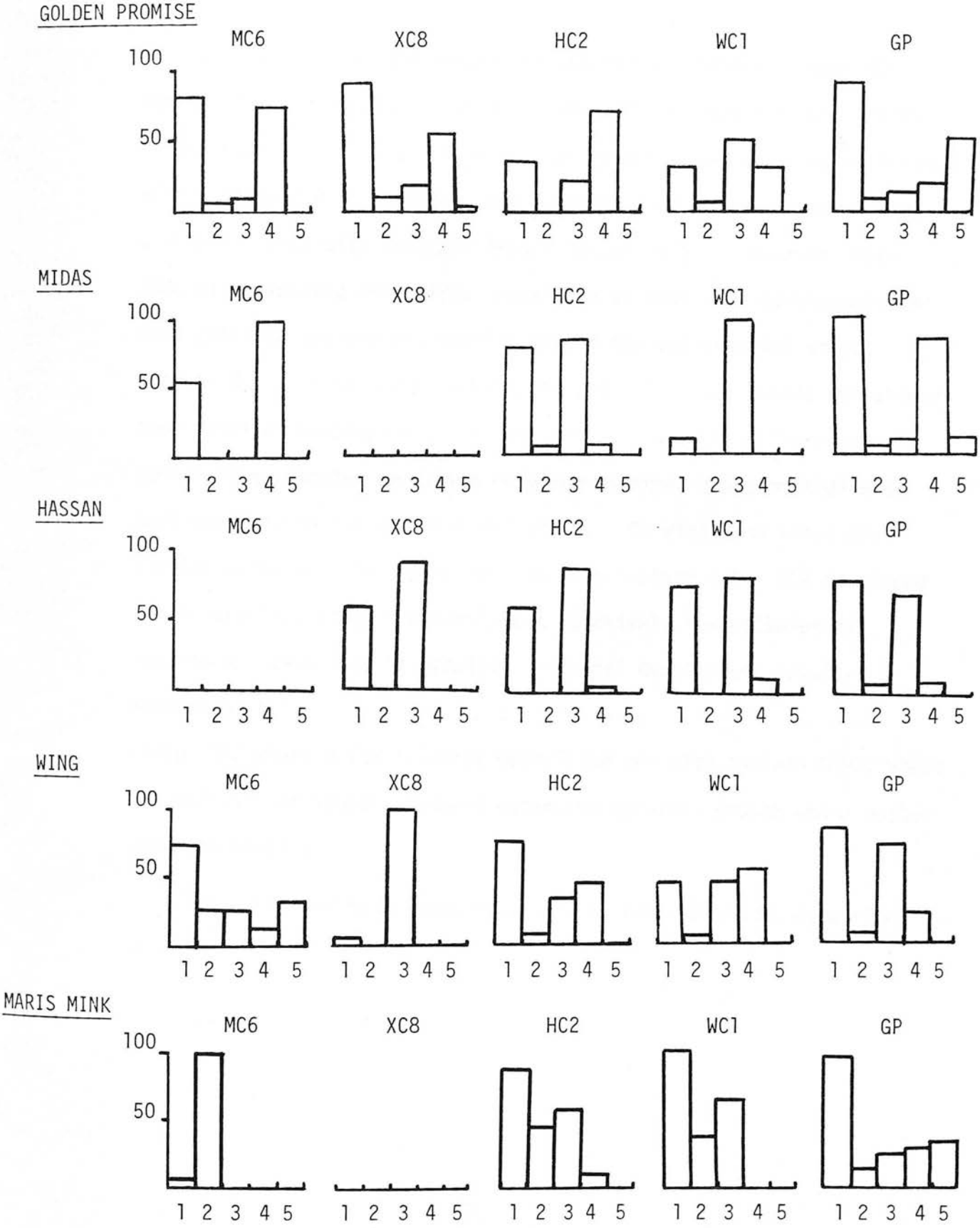


1 Germinated spores (total); 2 Germ tubes only present; 3 Appressoria present; 4 Mycelium present; 5 Conidia present (2-5 represent percentages of germinated spores).

on both cultivars most germinated spores had reached the appressorial stage. Germination rates for isolate HC2 on Golden Promise and Midas were poor. Race WC1 showed some germination on its compatible host Wing and germinated well on Hassan: again the majority of germinated spores were at the appressorial stage. On Golden Promise and Midas at 12 hours, no germinated spores of WC1 were found. At this assessment no race on any cultivar had developed beyond the stage of appressorial formation.

The results of the second assessment, 48 hours after inoculation, are summarized in Figure 14. At this stage isolate G.P. showed mycelial growth on all cultivars and sporulating colonies of this isolate were observed on segments of Golden Promise, Midas and Maris Mink. However, although there was some mycelial development on Hassan and Wing, the majority of spores on these cultivars had not developed beyond the appressorial stage. All isolates had developed mycelium on Golden Promise at 48 hours and a few colonies of races XC8 and MC6, as well as isolate G.P., had begun to sporulate. The only other race/cultivar combination to exhibit spore production was MC6 on Wing. All races had produced some mycelial colonies on Wing, except XC8 which had not developed beyond the appressorial stage. Isolates MC6 and G.P. produced numerous mycelial colonies on Midas but only a few spores of HC2 had developed mycelium and isolate XC8 had still failed to develop at all on this cultivar. The majority of spores inoculated onto Hassan had not developed beyond the appressorial stage although a few from races HC2 and WC1 were beginning to produce a mycelium; isolate MC6 still showed no development. On Maris Mink, isolate G.P. showed a proportion of spores which had undergone a full generation cycle but isolate HC2 was the only other isolate to develop

Figure 14. Development of Erysiphe graminis on leaf segments 48 hours after inoculation in relation to cultivar and isolate.



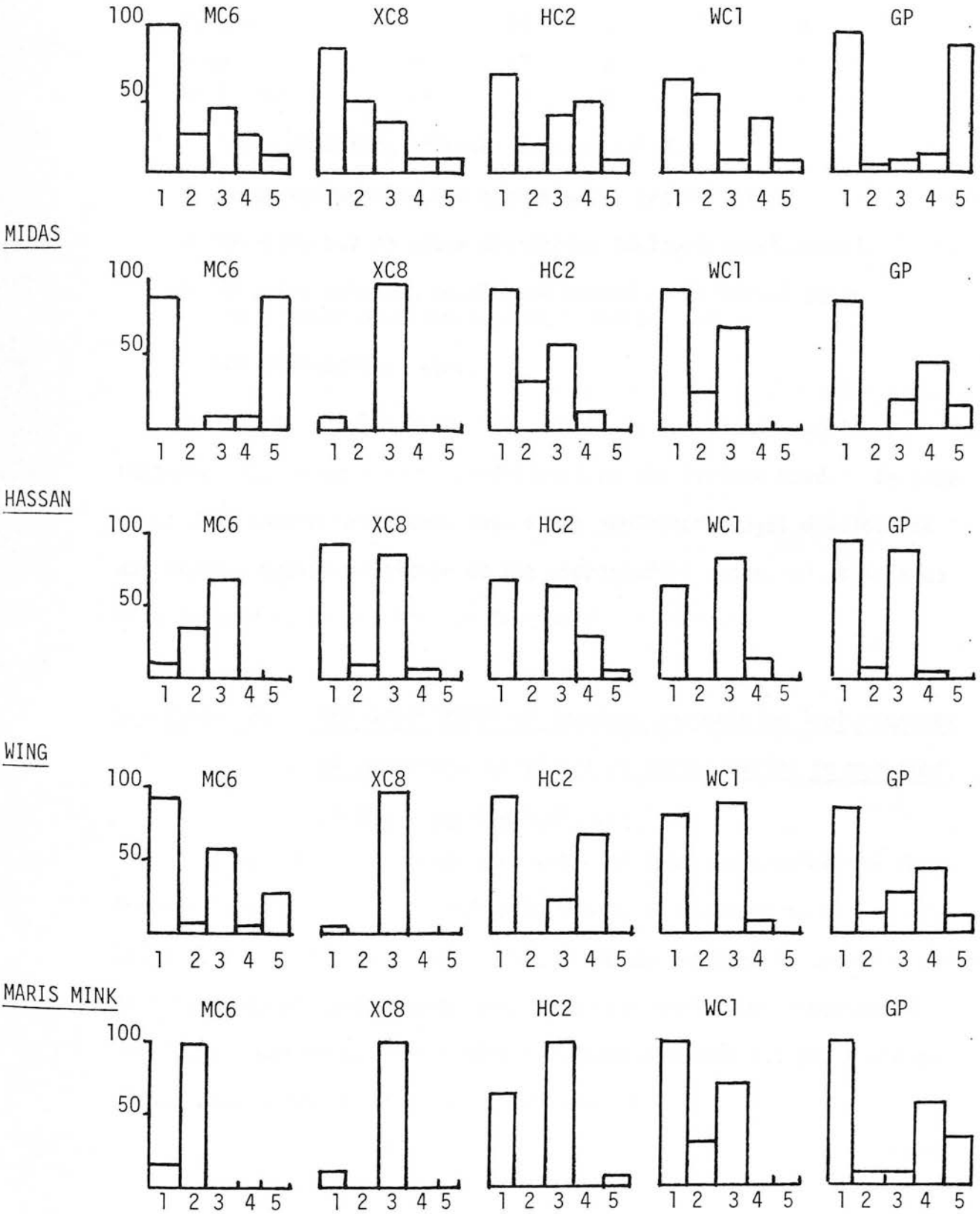
1 Germinated spores (total); 2 Germinated tubes only present; 3 Appressoria present; 4 Mycelium present; 5 Conidia present (2-5 represent percentages of germinated spores).

beyond the appressorial stage and isolate XC8 again showed no development.

Figure 15 shows the results of the final assessment made 108 hours after inoculation. At this time all isolates produced sporulating colonies on Golden Promise, the greatest number being associated with isolate G.P. On Midas, the percentage of colonies sporulating with MC6, originally isolated from Midas, was high. However, Race XC8, also isolated from Midas, continued to show poor development on this cultivar and did not develop beyond the appressorial stage; isolate WC1 also failed to develop beyond this stage, while HC2 showed some mycelial development. A few spores of isolate HC2 produced sporulating colonies on Hassan only and in other cases most development was only to the appressorial stage. On Wing some spore production occurred with MC6 as well as with isolate G.P: HC2 developed to the mycelial stage frequently but relatively few colonies of isolate WC1 gave rise to mycelium. Fungal development usually stopped at the appressorial stage on Maris Mink with all isolates except HC2 where a few colonies sporulated and with isolate G.P., where the majority of spores produced extensive mycelial growth and a number were sporulating.

The relationship between cultivar and isolate can be summarized as follows:-

Figure 15. Development of Erysiphe graminis on leaf segments 108 hours after inoculation in relation to cultivar and isolate.



1 Germinated spores (total); 2 Germ tubes only present; 3 Appressoria present; 4 Mycelium present; 5 Conidia present (2-5 represent percentages of germinated spores).

	Isolate				
Cultivar	MC6	XC8	HC2	WC1	GP
Golden Promise	+	+	+	+	+
Midas	+	-*	D	-	+
Hassan	-*	DR	+	D	D
Wing	+	-*	D	D	+
Maris Mink	-*	-*	R	-	+

+ Spore producing colonies (susceptible).

- No development beyond appressorial (resistant).

D Mycelium but no spore production (delayed development).

R Very few colonies developing beyond appressorial stage to produce mycelium or spores (restricted).

* Low germination rates.

Apart from the generally susceptible Golden Promise cultivar, responses may be seen to be conditioned by the isolate used. In some cases germination rates were low, while inhibited fungal development was usually exhibited either at the appressorial stage, or associated with delayed spore development from mycelial colonies.

Experiment 3b. The development of *Erysiphe graminis* on leaf segments of commercial cultivars of spring barley in relation to plant age and leaf position.

Cultivars for this study were selected from those which had shown intermediate levels of resistance to mildew in Experiments 2c and 2d. The development of fungal spores with time was studied in Experiment 3a: in this study all assessments were made five days after inoculation and account was taken of the effect of leaf position and plant age on fungal development as well as of cultivar effects.

Materials and methods.

Ten spring barley cultivars were used, covering a range of BMR groups with different resistance factors viz:-

Cultivar	Resistance Group	Resistance Factors
Proctor	0	-
Golden Promise	0	-
Zephyr	2	Mlg
Armelle	2	Mlg
Julia	2	Mlg
Imber	2	Mlg
Sultan	5	Mlas
Hassan	5	Mlas
Athos	2+5	Mlg+Mlas
Maris Mink	2+5+	Mlg+Mlas

Plants were grown in a spore free environment on an isolation propagator (Plates 4 and 5). When the plants were at the stem elongation stage (G.S. 31) standard 3 cm segments were taken from the top three of half the plants and transferred to benzimidazole agar in repli-dishes. Each cultivar was inoculated with spores of *E. graminis* produced on plants of that cultivar, using a camel-hair brush. Three segments were used for each leaf position and cultivar. The plates were incubated at 20°C, 16 hours daylength, for five days when the segments were cleared and stained for microscopic examination. The procedure was repeated for the remaining half of the plants when they were at the later "early booting" stage (G.S. 41). For each leaf position and cultivar at both growth stages between 100 and 200 spores were counted and put into one or other of the following five categories:-

1. ungerminated spore
2. germinated spore
3. appressorium formation
4. mycelial proliferation
5. sporulating colony.

These stages of development are illustrated in Plates 6 to 11.

Results.

The cultivars may be considered in three groups:-

A - Golden Promise, Proctor BMR0
B - Armelle, Julia, Imber, Zephyr BMR2
C { Hassan, Sultan BMR5
{ Athos, Maris Mink BMR2+5

Group A.

In the case of Golden Promise and Proctor (Table 35) the proportion of spores which germinated was fairly low.

Table 35. Percentage spores of E. graminis in different infection categories 5 days after inoculation onto Golden Promise and Proctor segments.

Harvest Date	Cultivar	Leaf Position	Infection Category * 1	2	3	4	5
11 April (GS 31)	Golden Promise	1 (top)	56	2	39	0	59
		2	78	1	78	1	20
		3	34	0	76	3	21
		Mean	56	1	65	1	33
11 April (GS 31)	Proctor	1	46	0	59	4	37
		2	46	2	72	2	24
		3	44	0	80	2	18
		Mean	45	1	70	3	26
30 April (GS 41)	Golden Promise	1	35	0	71	6	23
		2	43	2	77	2	19
		3	35	0	60	3	37
		4	33	0	76	3	21
		Mean	37	1	71	3	25
30 April (GS 41)	Proctor	1	39	0	85	5	10
		2	36	0	50	0	50
		3	39	3	51	3	43
		4	39	0	36	0	64
		Mean	38	0	55	3	42

* 1. Germinated spores.
2. Germ tubes present.
3. Appressoria present.
4. Mycelium present.
5. Sporulating colony.

} represented as a percentage of the germinated spores.

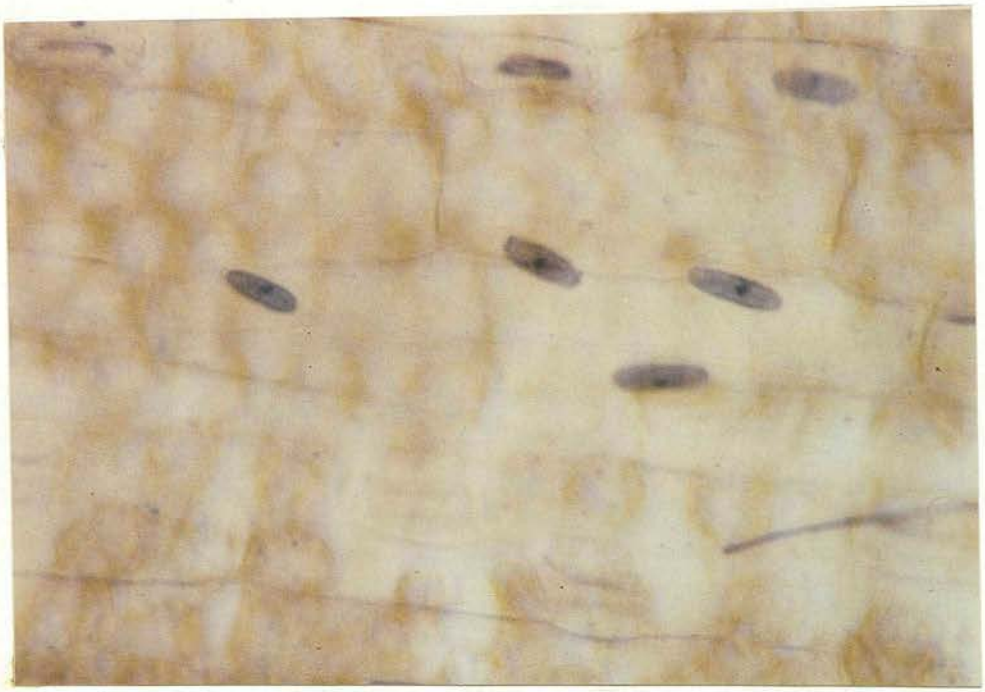


Plate 6. Ungerminated spores.

Plate 7. Formation of appressoria.

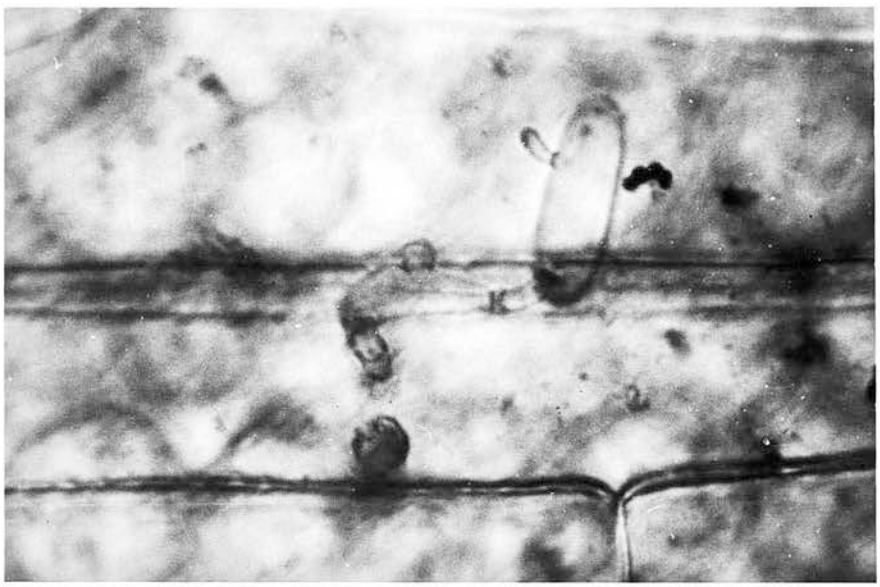


Plate 8. Formation of appressoria and growth of secondary germ tubes.

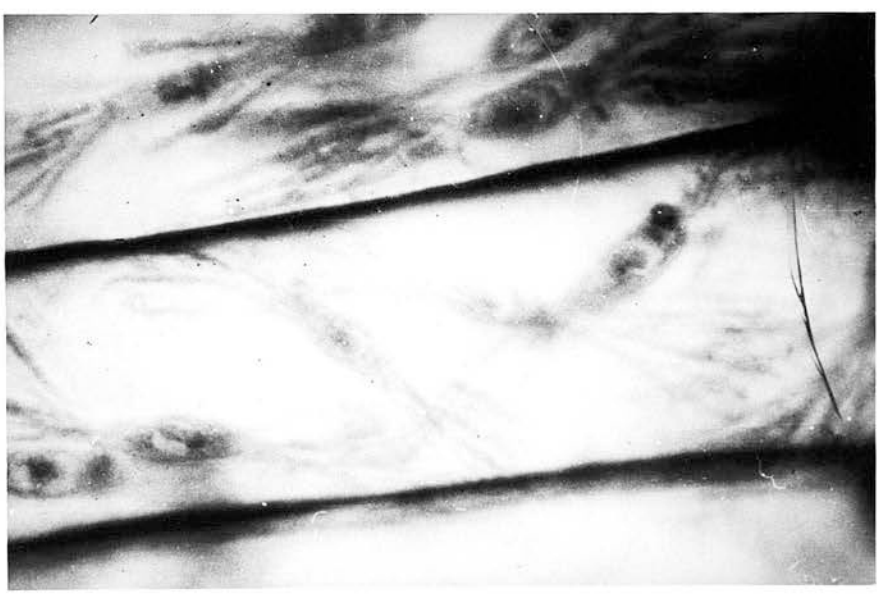


Plate 9. Establishment of haustoria.

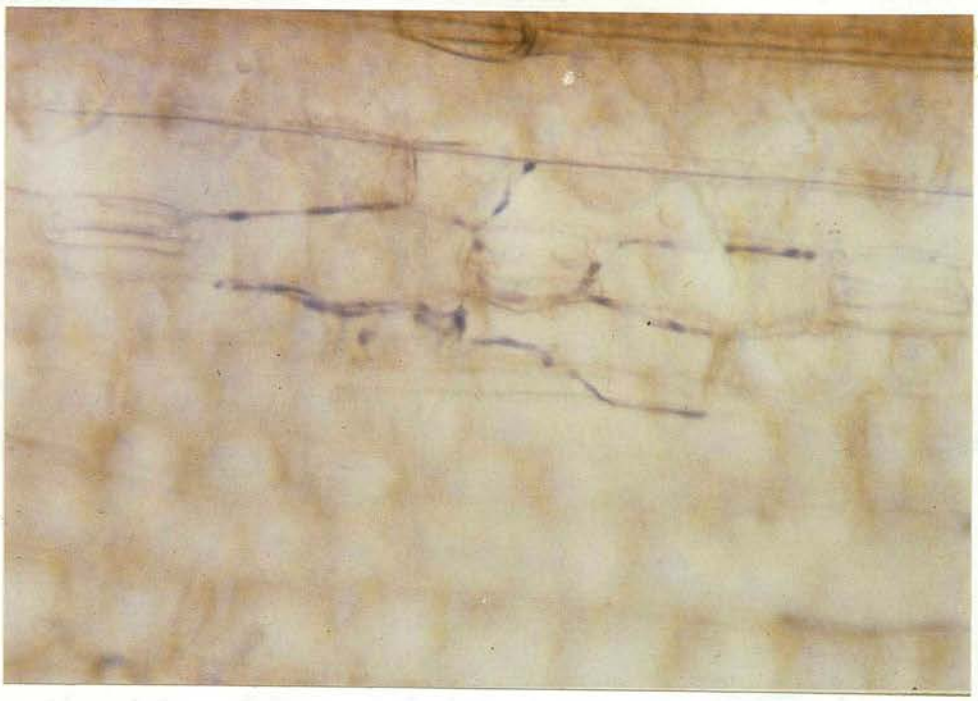


Plate 10. Proliferation of mycelium.

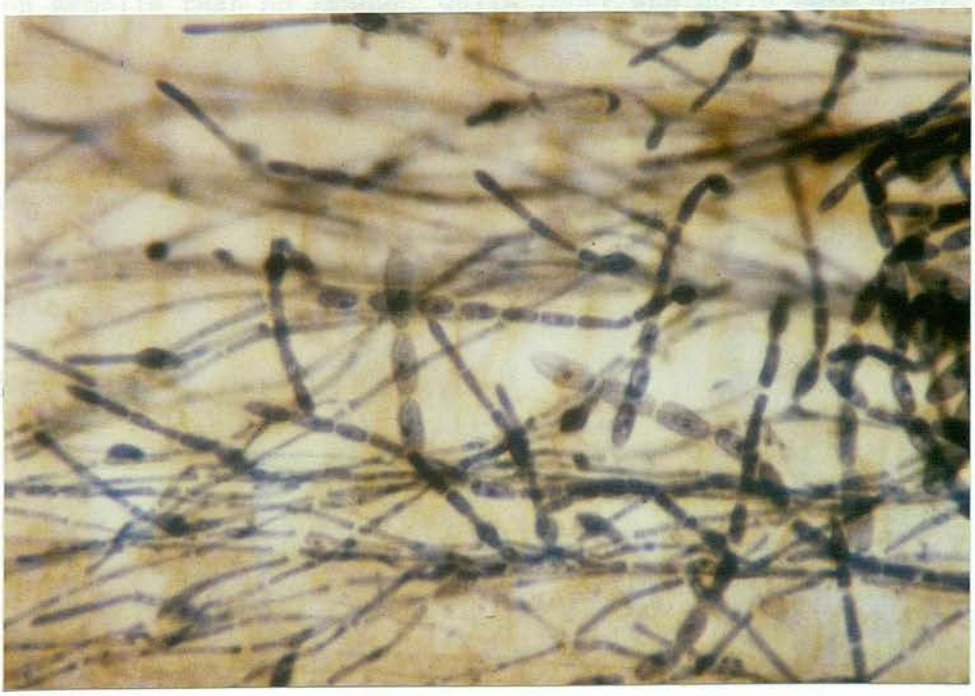


Plate 11. Development of conidia.

A relatively high preportion of those spores which did germinate produced sporulating colonies: the largest proportion, however, did not develop beyond the appressorial stage. No consistent pattern of development relating to leaf position, plant age or cultivar was seen. At the earlier growth stage more spores developed to produce sporulating colonies on the upper rather than the lower leaves; this was particularly marked with Golden Promise. At the later growth stage in the case of Proctor, there was a marked decrease in the numbers of sporulating colonies on the upper most leaf.

Group B.

On Armelle, Julia, Imber and Zephyr there was, overall, a lower percentage of spores which formed sporulating colonies in comparison with Group A plants. (Table 36). The total percentage of germinated spores was, however, higher. At the earlier growth stage (G.S. 31) fewer germinated spores had produced new sporulating colonies on Armelle than on Zephyr, while Imber and Julia were intermediate. At early booting stage (G.S. 41) the relative positions of Armelle and Zephyr were reversed, with Zephyr showing very few sporulating colonies. Imber and Julia were again intermediate. With regard to leaf position, at the earlier growth stage older leaves generally had fewersporulating pustules than the younger leaves but the reverse was true at G.S. 41.

Table 36. Percentage of spores of *E. graminis* in different infection categories 5 days after inoculation on Armelle, Julia, Imber and Zephyr.

Harvest Date	Cultivar	Leaf Position	Infection Category * 1	2	3	4	5
11 April (GS 31)	Armelle	1(top)	74	0	80	8	12
		2	72	0	90	6	4
		3	76	0	84	4	12
		Mean	74	0	85	6	9
	Imber	1	75	0	76	4	20
		2	83	1	90	3	6
		3	87	0	87	4	9
		Mean	82	<1	84	4	12
	Julia	1	55	2	63	2	33
		2	82	1	76	4	19
		3	70	1	83	4	12
		Mean	69	1	74	3	22
	Zephyr	1	37	3	62	5	30
		2	41	0	66	5	29
		3	54	2	63	11	24
		Mean	44	2	63	7	28
30 April (GS41)	Armelle	1	57	4	72	3	21
		2	38	0	50	0	50
		3	32	3	41	0	56
		4	40	0	77	0	23
		Mean	42	2	60	1	37
	Imber	1	66	2	97	1	0
		2	34	0	94	3	3
		3	40	0	80	2	18
		4	38	3	71	0	26
		Mean	45	1	85	2	12
	Julia	1	66	3	92	3	2
		2	63	2	94	4	0
		3	62	2	84	3	11
		4	24	0	21	8	71
		Mean	54	2	73	4	21
	Zephyr	1	71	4	94	2	0
		2	47	2	98	0	0
		3	59	2	97	0	1
		4	44	2	91	2	5
		Mean	55	3	95	1	1

* 1. Germinated spores.
2. Germ tubes present. } represented as a percentage
3. Appressoria present. } of the germinated spores.
4. Mycelium present. }
5. Sporulating colony. }

Group C.

Table 37 summarizes the results for Hassan and Sultan (BMR5), Athos (BMR 2+5) and Maris Mink (BMR 2+5+). Germination rates were low and the majority of germinated spores failed to develop beyond the appressorial stage. At G.S. 31 the germination rate was particularly low on Athos: for all four cultivars the percentage of germinated spores giving rise to sporulating colonies was between 12 and 20. At G.S. 41 germination was least on Hassan; low rates of spore production were associated with this cultivar and Athos. As with Group B, the effects of leaf position were reversed for the two plant ages: with young plants, young leaves showed more sporulating colonies, while at G.S. 41 older leaves showed greater fungal development.

Experiment 3c. Further studies on *Erysiphe graminis* on leaf segments of commercial cultivars of spring barley in relation to plant age and leaf position.

Experiment 3b was continued in this experiment, to examine further the influence of plant age, leaf position and their interaction with cultivar, on development of mildew. Four cultivars were used; Armelle and Zephyr (BMR2, Mlg) and Hassan and Sultan (BMR5, Mlas).

Materials and methods.

In the first part of this study plants were grown in spore free conditions on an isolation propagator until 8 weeks after sowing when they were at an average growth stage of 39. Half the plants were then removed for assessment and the others were grown on to an average growth stage 51. At each assessment date 3 cm segments were cut from the top

Table 37. Percentage spores of *E. graminis* in each infection category 5 days after inoculation on Hassan, Sultan, Maris Mink and Athos.

Harvest Date	Cultivar	Leaf Position	Infection Category * 1	2	3	4	5
11 April (GS 31)	Hassan	1(top)	38	3	50	5	42
		2	47	2	87	2	9
		3	50	0	88	4	8
		Mean	45	2	75	4	20
	Sultan	1	49	0	78	2	20
		2	64	0	86	2	12
		3	69	0	91	4	5
		Mean	61	0	85	3	12
	Maris Mink	1	53	0	68	11	21
		2	59	2	88	2	8
		3	17	0	88	6	6
		Mean	43	1	81	6	12
	Athos	1	43	0	63	9	28
		2	18	0	83	6	11
		3	20	5	80	5	10
		Mean	27	2	75	7	16
30 April (GS 41)	Hassan	1	63	2	95	3	0
		2	17	0	82	12	6
		3	32	3	91	3	3
		4	18	0	72	6	22
		Mean	32	1	85	6	8
	Sultan	1	61	0	75	3	22
		2	50	2	82	6	10
		3	41	0	54	7	39
		4	39	0	56	3	41
		Mean	48	1	66	5	28
	Maris Mink	1	60	2	93	3	2
		2	54	4	94	2	0
		3	59	2	76	3	19
		4	43	0	63	2	35
		Mean	54	2	81	2	15
	Athos	1	46	2	96	2	0
		2	57	3	93	2	2
		3	74	1	92	1	6
		4	40	2	78	2	18
		Mean	54	2	90	2	6

* for notation see Table 35.

four fully expanded leaves, inoculated with spores from infected plants of the same cultivar and incubated under the same conditions as in the previous experiment. The twelve replicates were laid out in a randomized block design. After four days incubation, half the segments were removed and prepared for microscopic assessment; after a further three days the remaining segments were assessed visually.

In the second part of the study seeds of the same four cultivars were sown on six sowing dates, at weekly intervals, in an isolation propagator. All plants were removed when the last sown plants were at G.S. 32. Standard 3 cm segments were taken from the top four fully expanded leaves of all plants and each cultivar was inoculated with an isolate of *E. graminis* taken from the same cultivar. One set of segments was incubated for 4 days and then assessed microscopically: a second set was incubated for 6 days and assessed visually. In this study four replicates of each cultivar, sowing date and leaf position combination were used and the experiment was arrayed in a randomized block design.

Results.

The results of the visual assessments are summarized in Table 38.

Table 38. Percentage surface area of leaf segments affected by mildew in relation to cultivar and leaf position.

Harvest date	Cultivar	Growth stage	Mildew percentage, leaf position 1	2	3	4	Mean
27 April	Armelle	35	6	7	16	22	13
	Zephyr	43	1	1	2	5	2
	Hassan	41	<1	2	2	7	3
	Sultan	37	1	3	4	8	4
	Mean	39	2	3	6	10	5
11 May	Armelle	50	0	<1	4	30	9
	Zephyr	54	<1	<1	4	12	4
	Hassan	50	3	3	3	-	3*
	Sultan	49	1	1	1	2	1
	Mean	51	1	1	3	11	4

* From upper three leaves only.

At both assessments younger leaves showed less infection: Armelle tended to show more infection than other cultivars.

The results of the microscopic analysis are given in Table 39 and summarized according to leaf position and cultivar in Tables 40a and 40b.

Table 39. Percentage of spores of Erysiphe graminis in different infection categories 4 days after inoculation in relation to cultivar and leaf position.

Harvest Date	Leaf Position	Cultivar	Infection Category * 1	2	3	4	5
27 April	1	Armelle	84	24	56	20	0
		Zephyr	77	32	68	0	0
		Hassan	84	43	52	5	0
		Sultan	82	51	33	16	0
	2	Armelle	82	29	56	15	0
		Zephyr	67	31	66	3	0
		Hassan	87	32	51	16	0
		Sultan	76	38	46	16	0
	3	Armelle	78	21	54	24	1
		Zephyr	77	13	56	31	0
		Hassan	88	34	55	11	0
		Sultan	86	43	35	22	0
	4	Armelle	76	24	58	17	1
		Zephyr	75	31	36	29	4
		Hassan	88	21	45	31	3
		Sultan	87	32	51	17	0
11 May	1	Armelle	63	48	51	1	0
		Zephyr	54	41	59	0	0
		Hassan	76	42	55	3	0
		Sultan	82	35	62	3	0
	2	Armelle	67	42	52	6	0
		Zephyr	72	40	52	8	0
		Hassan	79	37	48	14	1
		Sultan	78	45	50	4	1
	3	Armelle	70	40	47	13	0
		Zephyr	68	27	40	29	4
		Hassan	85	27	50	16	7
		Sultan	87	36	42	19	3
	4	Armelle	78	32	37	22	9
		Zephyr	64	34	27	36	3
		Hassan	86	22	32	37	9
		Sultan	90	17	59	23	1

* for notation see Table 35.

As with visual assessments a consistent effect of leaf position was found with younger leaves showing less infection (Table 40a).

Table 40a. Percentage of spores of Erysiphe graminis in different infection categories 4 days after inoculation in relation to leaf position.

Harvest Date	Leaf Position	Infection Category * 1	2	3	4	5
27 April	1	82	38	52	10	0
	2	78	32	55	13	0
	3	82	28	50	22	<1
	4	82	27	47	24	2
11 May	1	69	41	57	2	0
	2	74	41	50	8	1
	3	78	32	40	19	4
	4	80	26	39	29	6

* for notation see Table 35.

The development of *E. graminis* on different cultivars was generally at a similar level (Table 40b).

Table 40b. Percentage spores of Erysiphe graminis in different infection categories 4 days after inoculation in relation to cultivar.

Harvest Date	Cultivar	Infection Category * 1	2	3	4	5
27 April	Armelle	80	24	56	19	1
	Zephyr	74	27	56	16	1
	Hassan	87	32	51	16	1
	Sultan	83	41	41	18	0
11 May	Armelle	70	41	47	10	2
	Zephyr	65	26	41	18	2
	Hassan	82	31	46	18	4
	Sultan	84	33	53	12	1

* for notation see Table 35.

Table 41. Percentage surface area of leaf segments affected by mildew in relation to cultivar and leaf position.

Sowing Date	Cultivar	Growth Stage	Mildew Percentage				
			Leaf Position				
			1	2	3	4	Mean
27 Feb	Armelle	49	0	0	0	-	0
	Zephyr	54	0	0	0	-	0
	Hassan	56	0	0	5	-	2
	Sultan	55	0	0	0	-	0
	Mean	54	0	0	1	0	<1
6 Mar	Armelle	41	1	<1	2	0	1
	Zephyr	54	<1	0	<1	<1	<1
	Hassan	49	0	1	1	1	1
	Sultan	48	0	0	0	1	<1
	Mean	48	<1	<1	1	1	1
13 Mar	Armelle	33	8	10	12	18	12
	Zephyr	43	3	4	13	26	12
	Hassan	42	3	3	11	27	11
	Sultan	37	20	4	15	20	15
	Mean	39	8	5	13	23	12
20 Mar	Armelle	34	15	17	19	22	18
	Zephyr	36	12	18	9	28	17
	Hassan	36	5	10	21	8	11
	Sultan	35	8	21	7	30	17
	Mean	35	10	16	14	22	16
27 Mar	Armelle	32	15	5	2	23	11
	Zephyr	34	3	5	23	19	13
	Hassan	33	10	11	6	10	9
	Sultan	34	9	17	28	36	23
	Mean	33	9	9	15	22	14
3 April	Armelle	37	17	11	6	5	10
	Zephyr	33	3	3	9	13	7
	Hassan	33	17	10	12	27	17
	Sultan	32	13	20	16	20	17
	Mean	32	13	11	11	16	13

The results of visual assessments in the second study are summarized in Table 41. All plants from the earliest two sowing dates showed negligible amounts of infection: for the remaining plants the oldest leaves were the most severely infected. In this experiment there was no marked difference between Armelle and Zephyr; in comparing Sultan and Hassan, however, Hassan tended to show less infection, particularly on the lower leaves at stem elongation. The results of the microscopic observations are given in relation to leaf position (Table 42a) and cultivar and growth stage (Table 42b).

Table 42a Percentage of spores of Erysiphe graminis in different infection categories 4 days after inoculation in relation to leaf position.

(Mean of cultivars)

Sowing Date	Growth Stage	Leaf Position	Infection Category * 1	2	3	4	5
27 Feb	54	1	84	20	74	6	<1
		2	80	18	75	7	0
		3	80	12	84	4	0
		4	-	-	-	-	-
6 Mar	48	1	92	12	87	1	0
		2	87	14	85	1	0
		3	80	15	82	3	0
		4	94	10	85	5	0
13 Mar	39	1	89	12	81	7	<1
		2	87	11	80	9	0
		3	89	11	76	13	0
		4	93	10	63	26	1
20 Mar	35	1	87	12	75	12	1
		2	91	8	75	16	1
		3	87	11	70	18	1
		4	90	6	66	27	1
27 Mar	33	1	89	9	75	14	2
		2	91	8	75	16	1
		3	90	9	68	22	1
		4	91	9	65	25	1
3 April	32	1	90	10	80	10	<1
		2	92	12	73	14	1
		3	89	8	73	17	2
		4	89	7	74	19	0

* for notation see Table 35.

Table 42b. Percentage of spores of Erysiphe graminis in different infection categories 4 days after inoculation in relation to cultivar and growth stage.

(Mean of upper four leaves)

Sowing Date	Cultivar	Growth Stage	Infection Category * 1	2	3	4	5
27 Feb	Armelle	49	71	19	78	3	<1
	Zephyr	54	83	19	80	1	0
	Hassan	56	88	16	79	5	0
	Sultan	55	87	17	70	13	0
	Mean	54	82	18	77	6	<1
6 Mar	Armelle	41	93	11	87	3	0
	Zephyr	54	83	15	84	1	<1
	Hassan	49	81	18	80	2	0
	Sultan	48	88	11	86	3	0
	Mean	48	86	14	84	2	<1
13 Mar	Armelle	33	94	15	74	11	<1
	Zephyr	43	86	7	75	18	<1
	Hassan	42	89	10	74	15	1
	Sultan	37	89	11	78	11	0
	Mean	39	89	11	75	14	<1
20 Mar	Armelle	34	91	9	74	17	1
	Zephyr	36	87	12	75	12	1
	Hassan	36	90	8	62	29	1
	Sultan	35	87	11	75	14	<1
	Mean	35	89	10	72	18	1
27 Mar	Armelle	32	92	9	74	16	1
	Zephyr	34	91	7	75	17	1
	Hassan	33	85	7	68	23	2
	Sultan	34	93	12	65	22	1
	Mean	33	90	9	71	20	1
3 April	Armelle	31	94	10	75	15	<1
	Zephyr	33	86	7	81	11	1
	Hassan	33	85	8	73	17	2
	Sultan	22	93	12	70	17	1
	Mean	32	89	9	75	15	1

* for notation see Table 35.

A large number of spores inoculated onto older plants (G.S. 48 and 54 at harvesting) failed to develop beyond the appressorial stage and more advanced fungal development was generally seen on younger plants especially on the older, lower leaves. There were no consistent differences between cultivars as regards mildew development.

DISCUSSION.

In the first experimental sections, studies were based on polycyclic testing where plants were exposed to recurrent infection cycles during their growth and development on the host. These studies pointed to a range of host responses some of which reflected major gene interactions while others expressed levels of partial resistance. The effects of major genes seemed to operate soon after penetration (Ellingboe, 1972; Koga, Mayama and Shishiyama, 1978), whereas other mechanisms still concerned with tissue resistance may operate at other stages (Hyde and Colhoun, 1975).

The experiments in the third section aimed to examine further the nature of host-parasite interactions, especially in relation to partial resistance, by monocyclic testing (Zadoks, 1972) when components of pathogen development in a single infection cycle were considered.

This third set of investigations looked at responses where major genes were involved and reaction types tended to be highly susceptible or highly resistant. In susceptible interactions colony development was rapid and in some cases the generation time was as little as 48 hours. The extent to which this occurred varied with isolate and host. By 108 hours after inoculation all susceptible combinations had reached the spore production stage. Incompatible combinations generally showed little development beyond the appressorial stage although there were differences and an occasional host/pathogen combination produced a sporulating colony. Ellingboe (1972) observed that a limited number of parasitic units of wheat and barley mildew always reached maximum development regardless of the host-parasite genotypes. Conversely, it was observed in the present work that even with highly susceptible

responses some spores failed to develop past the appressorial stage. Variation in individual spore development behaviour was therefore shown in all forms of resistance/susceptible responses. In some cases spore behaviour may be linked with the physiological condition of the spore (Ellingboe, 1972); in others the response of individual host cells may vary (Koga *et al*, 1978). Lupton (1956) observed that the behaviour of several susceptible lines to wheat mildew differed widely with inoculation conditions and differences in response were often exhibited by different parts of the same leaf.

It was recognised in the present microscopic work that spore germination was at times poor in spite of using spores no more than 24 hours old (Ellingboe, 1972). From the findings of Russell, Andrews and Bishop, (1975) they indicated that germination of barley mildew conidia may be affected by the host genotype and growth stage, as well as by the area of the leaf concerned.

In the first study of this experimental section (Experiment 3a) spore germination rates were found to vary with isolate and cultivar: all isolates showed the greatest germination rates on Golden Promise and the isolates cultivated from Golden Promise germinated most successfully of all isolates on any host. Later experiments did not confirm this, however, and germination rates appeared to be an expression of the cultivar/isolate combination rather than either factor alone. No clear pattern emerged from a consideration of the influence of growth stage and leaf position on spore germination, although younger leaves were sometimes associated with higher germination rates. Lupton (1956) working on wheat mildew considered that germination was not affected by the host and Ellingboe (1972) considered that incompatible host responses occurred after penetration. However, Russell *et al* (1975) did not preclude some quantitative variation in spore germination being linked

with partial or adult plant resistance.

In the second and third studies, account was taken of plant age and leaf position on mildew development. A general observation was that from stem elongation onwards the younger functional leaves showed a more limited development of germinated spores than did older leaves. An exception to this was Golden Promise which showed a similar development on all leaves; Proctor, in comparison, conformed to the general pattern of spore development, a result in keeping with the observations of the polycyclic test in Experimental Section 1. There was no similar correlation between the findings of the monocyclic and polycyclic tests on those cultivars with Mlg resistance. Thus Armelle tended to show a higher level of partial resistance than Zephyr in tests on disease development in the glasshouse but no delayed spore development in microscopic tests. In comparing Sultan and Hassan, both with Mlas resistance, Hassan showed poorer spore development at the later growth stage, confirming earlier observations. There was no obvious effect of the combination of Mlg and Mlas compared with either factor alone. In the final microscopic studies with Armelle, Zephyr, Hassan and Sultan, Armelle again appeared to have no resistance advantages over Zephyr but Hassan showed poorer fungal development than Sultan.

Partial resistance indicated in glasshouse trials was often not confirmed in monocyclic studies. In microscopic tests inoculum was derived in all cases from the same cultivars as the inoculated host to ensure a compatible combination of major genes in host and parasite, thus any variation would not be a result of major gene effects. In contrast, in the glasshouse inoculum on all plants was by a population of mildew with no recognised genetic bias to any cultivar. Wolfe and Schwarzbach (1978) have observed that cultivars possessing Mlg resistance have shown a wide range of susceptibility to virulent populations

of the pathogen, possibly due to difference in background genes. The A.D.A.S. cereal foliar disease survey showed that the level of infection on Zephyr declined with the reduction in acreage under this cultivar, while infection levels on Julia increased with its greater cultivation. The failure of Armelle to show any genetic advantage over Zephyr in the present microscopic studies may have been related to each cultivar being inoculated with its specific pathogen genotype.

The findings of these studies were further qualified in that only certain components in the infection cycle which might have no epidemiological significance were considered. The progress of individual spores was examined with respect to the stage colony development achieved and as already indicated, some variation in this may be due to the physiological condition of the spore rather than simply the host genotype. Generally, very few spores germinated without producing appressoria, in keeping with the observations of Ellingboe (1972). In the present study most infection failure took place soon after the appressorial stage: when hyphae were produced at the infection site development usually progressed to spore production.

Other factors which could be considered in later work may be the latent period, sporulation rate and infectious period (Zadoks, 1972), all factors of epidemiological significance. A further factor which may determine infection level in the field is spore deposition rate. Russell (1975) indicated that the deposition rate of mildew spores on barley cultivars is influenced by growth habit and that an erectly growing plant may contribute to disease escape in the field.

EXPERIMENTAL STUDIES 4.

Studies on *Rhynchosporium secalis* of barley.

EXPERIMENTAL STUDIES 4.

Studies on *Rhynchosporium secalis* of barley.

Introduction.

In this section studies on *Rhynchosporium secalis* are described. Preliminary work had indicated that this pathogen was more demanding in its requirements for establishing infection and active growth than *Erysiphe graminis*: this contrasting behaviour of the two pathogens was illustrated by the observation that *E. graminis* often caused contamination in *R. secalis* experiments. With mildew, disease occurred readily, often from natural infection, with no environmental manipulation: in the case of *R. secalis*, however, it was necessary to provide a controlled environment with particular respect to the humidity which required to be maintained at near saturation. Experiments on *R. secalis* comprised a) an assessment of a range of barley lines and cultivars for their susceptibility to infection; b) leaf segment studies on variation in symptom development and spore production on a number of commercial cultivars and c) a study of yield responses to leaf blotch infection by commercial cultivars showing different levels of resistance.

Experiment 4a. Screening of barley seedlings for resistance to infection by *Rhynchosporium secalis*.

This experiment was designed to develop a method for the production of conditions favourable for leaf blotch development and to assess a range of barley lines and cultivars for their disease response when inoculated with *Rhynchosporium secalis*. For spore germination and appressorial development *R. secalis* requires free water on the leaf surface (Ayesu-Offei and Clare, 1970): wet conditions are necessary

for abundant production of conidia and splash dispersal may be important in the dissemination of propagules (Doling, 1964; Ayesu-Offei and Carter, 1971).

Preliminary attempts to establish infection, on whole plants in glasshouse compartments, had involved the use of open trays of water placed at intervals in the compartment. It was hoped that evaporation from the water surface would maintain the humidity at a high enough level to promote fungal development. This was not successful, however, and for the present work an automatic mist propagation system was installed in two glasshouse compartments. A leaf surface wetness sensor was used to control the amount of moisture on the leaves at any moment. In other experiments glass frames, fitted with manually operated misting equipment, were used. In the first investigation (a) using automatic misting, one race of *R. secalis* was used, while in the second (b) with manually controlled misting, two races were used.

Materials and methods.

(a) Entries from the European and Expanded European Collections (Appendix I) were sown in pairs of rows 0.15 m apart with 0.5 m spacing between each pair, in two 4.5 m square glasshouse compartments used as replicates. The compartments were each fitted with an automatic mist propagation system, controlled by a surface wetness sensor (Plate 12), which periodically exposed plants to a fine spray and maintained a film of water on the leaf surface.

Inoculum was prepared by harvesting spores from 14 day old cultures of U.K. Race 1 (Clifford and Jones, 1978) originally supplied by the Welsh Plant Breeding Station, Aberystwyth. The cultures were produced on lima bean agar and the spores made into suspensions of 2×10^6 spores/ml in sterile distilled water. Three litres of suspension

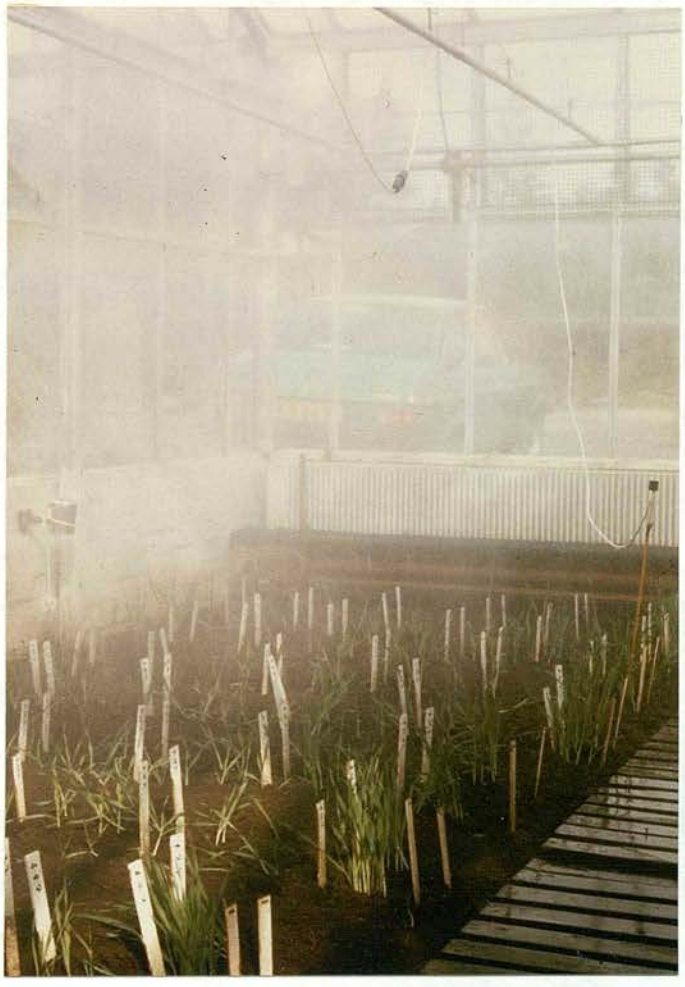


Plate 12. Automatic mist propagation system showing leaf surface wetness sensor.

were sprayed in each compartment when plants reached the 4-5 leaf stage. Plants of the susceptible cultivar Maris Mink were grown at the end of each row: these developed severe infection and helped to maintain inoculum throughout the period of growth. Assessments were carried out at intervals during the growing period by determining the percentage leaf area of the upper four fully expanded leaves affected by *R. secalis*. Lines and cultivars were placed in categories, based on the highest infection rating found over the period of observations, as follows:-

0 - no infection
1 - not more than 1 per cent leaf blotch
2 - " " " 10 " " " "
3 - " " " 25 " " " "
4 - " " " 50 " " " "
5 - over 50 per cent leaf blotch.

(b) A collection of commercial cultivars were grown in 5 cm fibre pots in a 2m x 1m x 1m glass frame fitted with a manually operated mist propagation system. The plants were sprayed with a fine mist of 30 minutes twice daily; this maintained a moist atmosphere in the cabinets. The plants were inoculated at G.S. 14 with a suspension of *R. secalis* (2×10^6 spores/ml) prepared as described previously: Races U.K.1 and U.K.2 were used and there were four cultivar replicates for each race. The plants were visually assessed for *R. secalis* infection two and six weeks after inoculation, by estimating the surface area affected.

Results.

(a) In the compartments with automatic misting apparatus, Maris Mink plants became severely infected. Results of the assessments of barley lines are summarized in Table 43.

Table 43. Development of Rhynchosporium secalis on plants of European Collections.

	Percentage lines in each infection category					
	0	1	2	3	4	5
European Barley Disease Nursery	2	0	21	28	45	4
Expanded European Barley Disease Nursery	7	0	20	41	31	1

The majority of lines were susceptible to *R. secalis* infection. There were, however, a few which showed no infection and about 20% of each collection gave relatively low levels of infection.

(b) The results of the assessment of commercial cultivars are given in Table 44. Spring barley cultivars at G.S.21 were more heavily infected than those at the later stage; Race U.K.1, however, caused more infection than Race U.K.2 at both growth stages. Winter barley cultivars were generally uninfected. Comparison with N.I.A.B. resistance ratings given show some inconsistency which may be a reflection of the difficulties of initiating an epidemic of *R. secalis*.

Table 44. Percentage Rhync hosporium secalis averaged over upper four leaves of commercial cultivars at two growth stages. (Races UK1 and UK2).

	Cultivar	Growth Stage				NIAB resistance rating *	
		21		35			
		UK1	UK2	UK1	UK2		
						1976/77	1982/83
Spring cultivars	Golden Promise	5	0	1	5	C	C
	Tyra	1	0	0	10	C	A
	Sundance	5	0	0	25	D	B
	Abacus	10	0	0	0	C	-
	Yamina	5	0	0	0	-	-
	Zephyr	25	0	0	0	D	-
	Georgie	75	0	0	0	C	B
	Goldmarker	5	0	0	0	-	B
	Porthos	-	0	1	0	D	B
	Midas	0	0	-	0	D	B
	Athos	25	0	0	0	C	B
	Dram	25	0	0	0	-	B
	Lami	25	0	0	5	D	-
	Katy	-	0	0	0	-	-
	Maris Mink	10	0	0	0	D	D
	Julia	25	0	0	0	D	-
	Minak	5	0	0	0	-	-
	Piccolo	5	0	0	0	-	D
	Triumph	-	0	0	-	-	B
	Ark Royal	25	0	10	0	C	A
	Goldspear	25	0	0	5	-	C
	Hassan	25	0	0	0	C	C
	Koru	5	0	10	0	-	A
	Havila	1	1	0	0	-	B
	Tintern	5	5	5	0	-	B
	Goblin	0	0	0	0	-	B
	Aurea	25	0	1	0	-	B
	Guilden	-	0	0	0	-	-
	Herzo	10	0	0	0	-	-
	Kite	50	0	5	0	-	-
	Polka	25	0	0	0	-	-
	Allegro	25	0	10	0	-	-
	Anta	25	0	0	0	-	-
Winter cultivars	Hoppel	0	0	0	0	A	-
	Athene	-	0	0	0	-	B
	Astrix	-	0	0	0	A	-
	Igri	-	0	0	0	-	A
	Sonja	0	0	5	0	A	B

* A = High level of resistance.

Experiment 4b. Leaf segment studies on the development of *Rhynchosporium secalis* on different barley cultivars.

The extent of colony development and associated spore production, in the case of *Erysiphe graminis* infection, can be related directly to the amount of disease observed in visual assessments. *Rhynchosporium secalis*, however, develops below the leaf surface and damage due to toxin and enzyme activity may occur at a distance from the actively growing fungus (Ayesu-Offei and Clare, 1971; Jones and Ayres, 1972). Visual disease assessment may not, therefore, directly correspond to fungal activity. More importantly the extent of spore production, which partly reflects the infection rate of the pathogen, may not be related to the visual disease index. With mildew, necrotic symptoms indicate a hypersensitive response reaction and an incompatible host/pathogen combination; with leaf blotch a visible breakdown of tissue is, however, an early consequence of successful infection.

In the present studies investigations were carried out on different barley cultivars with different levels of resistance to *R. secalis*, to assess visual symptom development and spore production on leaf segments inoculated with a Maris Mink isolate of *R. secalis*. Ayres and Owen (1970) have indicated that detached leaves perform in a similar way to whole plants.

Materials and methods.

Three series of studies were undertaken. In the first, 12 spring barley cultivars were studied; a further six were included in a second trial and winter barley cultivars were studied in the third experiment. For all studies plants were grown in growth cabinets with a 16 hour day at 15°C and removed for inoculation at G.S. 30-32.

The top four leaves of the main tiller plus the top leaf of the leading side tiller were taken and a 3 cm segment was removed from the mid-region of each leaf. The segments were placed on 80 ppm benzimidazole agar and inoculated with a suspension of *R. secalis* ($6x10^5$ spores/ml, using an Agla syringe to give a uniform droplet size (Kay and Owen, 1973). The inoculation segments were then incubated under the same conditions as the original plants and were visually assessed at 4-8 day intervals for symptom development using aneight point scale illustrated in Plate 13. At the final assessment, 29-30 days after inoculation, leaf segments were placed in a fixed volume of lacto-phenol cotton blue. The leaf tissue was teased out with mounted needles to release spores into the liquid. The concentration of spores released was assessed using a haemacytometer slide. There were eight or twelve replicates of leaf position and cultivar in visual assessments and four replicates in assessing spore production.

Results.

The results of the first experiment using 12 spring barley cultivars are summarized in Table 45. Only faint symptoms of infection were evident one week after infection; there was a progressive increase in the severity of symptom expression until the fourth week by which time the segments had become senescent. Symptom expression and spore development was least on the oldest leaf, i.e. leaf 4. The severity of expression varied with cultivar: Tyra was most severely infected, Armelle and Minak the least. Spore production did not show a close relationship with visual symptom assessment but Tyra, the most severely infected cultivar, also had a high spore production index. Akka and Keg showed very low levels of spore

Plate 13. Symptom severity scale for Rhynchosporium secalis infection.

from left to right:-

0 no symptoms
1 slight chlorosis
2 slight necrosis
3 slight chlorosis and necrosis
4 extensive chlorosis
5 extensive necrosis
6 almost complete lesion
7 complete lesion.

Table 45. Development of Rhynchosporium secalis on leaf segments of twelve spring barley cultivars.

	Symptom assessment (0-7, Plate 13)					Spore production index*
	Days after inoculation					
	8	12	19	23	30	30
Leaf number (mean of all cultivars)						
1(top)	0.0	0.3	3.3	4.5	4.7	9.7
2	0.7	0.7	2.9	4.4	4.7	14.2
3	0.6	0.8	2.4	2.9	3.2	9.0
4	0.3	0.3	0.7	1.2	1.5	3.2
1(tiller)	0.4	0.1	1.0	2.2	2.3	7.2
Cultivar (mean of leaf positions)						
Tyra	0.8	1.1	3.6	4.9	5.2	16.6
Akka	0.4	0.5	2.7	4.2	5.0	1.5
Piccolo	0.4	0.2	2.1	3.9	4.0	7.0
Keg	0.4	0.6	2.7	3.9	3.7	1.8
Hassan	0.3	0.4	1.7	3.3	3.5	7.3
Berac	0.1	0.3	2.3	3.2	3.3	19.6
Imber	0.3	0.3	1.5	3.0	3.3	7.8
Dram	0.4	0.6	2.2	2.6	2.7	9.6
Mala Abed	0.3	0.5	2.1	2.5	2.6	7.6
Athos	0.3	0.4	1.3	2.1	2.1	15.3
Armelle	0.0	0.1	0.6	1.4	2.0	5.7
Minak	0.1	0.4	1.8	1.9	2.0	4.1
S.E.D.±	0.22	0.24	0.60	0.56	0.58	3.79
(D.F.)	(109)	(110)	(49)	(50)	(50)	(20)

* spore/ml sample.

production although they were among cultivars showing more severe symptoms. In contrast, Berac and Athos showed high rates of spore production relative to their symptom severity rating.

The results of work on six cultivars in the second study are summarized in Table 46. Disease levels were generally lower, as assessed by visual symptom expression, than in the previous study although spore production indices were of a similar order. As in the previous experiment, infection ratings of the main tiller were greatest on the second leaf and least on the lowest leaf. Tyra was again the highest ranking in order of symptom severity, Georgie the least. Differences in spore production between cultivars were not significant.

Table 46. Development of Rhynchosporium secalis on leaf segments of six spring barley cultivars.

	Symptom assessment (0-7, Plate 13)					Spore production index*
	Days after inoculation					
	8	12	19	23	30	30
Leaf number (mean of all cultivars)						
1(top)	0.1	0.3	1.6	2.3	2.7	8.2
2	0.3	0.6	0.9	1.6	2.1	18.3
3	0.0	0.0	0.2	0.5	0.5	3.3
4	0.1	0.1	0.1	0.3	0.3	0.3
1(tiller)	0.0	0.1	0.6	2.6	2.7	7.4
Cultivar (mean of leaf positions)						
Tyra	0.3	0.3	0.9	1.9	2.4	5.5
Maris Mink	0.1	0.3	1.0	2.0	2.1	3.8
Armelle	0.0	0.0	0.8	1.8	1.9	12.6
Sundance	0.2	0.2	0.3	0.8	1.3	8.8
Proctor	0.0	0.2	0.5	1.3	1.3	7.2
Georgie	0.0	0.3	0.7	0.9	0.9	6.8
S.E.D,±	0.12	0.16	0.32	0.53	0.59	6.22
(D.F.)	(121)	(121)	(66)	(63)	(63)	(8)

* spore/ml sample.

The third study on winter barley cultivars and Maris Mink again showed a leaf position effect (Table 47) with the fourth leaf showing least infection. Astrix showed severe symptoms and had a high level of spore production compared with other cultivars.

Experiment 4c. Susceptibility of spring barley cultivars to infection by *Rhynchosporium secalis* and yield relationships.

Hapgood (1975) found that in field conditions different cultivars could show appreciable differences in infection levels and infection rates but the relevance of this to yield loss was not discussed. Previously, Schaller (1963) reported that yield losses due to *R. secalis* infection was the result of a fall in the number of grains per ear. However, James, Jenkins and Jennett (1968) reported that yield losses were due to a reduction of thousand grain weight. Rowlings and Jones (1976) attributed this discrepancy to the different timings of the epidemics in relation to plant growth stage. They found a significant yield loss in the susceptible cultivar Mosane in response to a single inoculation, whilst the more resistant Proctor required multiple inoculations before any adverse effect was recorded. Symptom expression in Vada and Mosane was not significantly different, but yield losses were less in Vada than Mosane. This study was aimed to assess the relative susceptibility of spring barley cultivars to infection by two races of *R. secalis* and to determine the effects of infection on yield components.

Materials and methods.

seven spring barley cultivars were used representing a range of NIAB resistance ratings (Anon, 1979):-

Table 47. Development of Rhynchosporium secalis on leaf segments of winter barley cultivars.

	Symptom assessment (0-7, Plate 13)					Spore production index *
	days after inoculation					
	8	12	19	23	30	30
Leaf number (mean of all cultivars)						
1(top)	0.0	0.1	0.8	3.0	4.0	20.7
2	0.2	0.3	0.5	2.4	3.4	15.0
3	0.3	0.3	0.3	1.3	1.8	15.9
4	0.1	0.0	0.2	1.2	1.7	7.9
1(tiller)	0.0	0.0	0.6	2.5	3.1	15.9
Cultivar (mean of leaf positions)						
Astrix	0.1	0.1	0.4	2.8	3.7	27.7
Hoppel	0.1	0.1	0.4	1.9	3.4	17.8
Athene	0.1	0.1	0.4	2.4	3.0	23.4
Maris Otter	0.1	0.1	0.6	2.0	2.7	6.7
Sonja	0.0	0.3	0.4	1.8	2.3	7.8
Maris Mink	0.3	0.2	0.6	1.7	1.8	7.0
S.E.D.±	0.12	0.16	0.32	0.53	0.59	8.79
(D.F.)	(121)	(121)	(66)	(63)	(63)	(12)

* spore/ml sample.

Cultivar	Resistance (0 = susceptible) (9 = resistance)
Armelle	7
Proctor	6
Dram	5
Minak	5
Vada	3
Golden Promise	3
Maris Mink	1

These plants were grown in two 4.5 m square glasshouse compartments, each fitted with automatic mist propagation equipment to provide a damp atmosphere (Experiment 4a). Ten plants of each cultivar were grown in a 25 cm plant pot; Tyra plants were used as guard rows.

Spore suspensions were made in the usual way and applied as a fine mist to run-off point in four inoculation treatments; control (uninoculated); early inoculation (7 weeks after sowing); late inoculation (10 weeks after sowing); and a double inoculation treatment carried out at both dates. Race U.K.1 was used in one compartment and Race U.K.2 in the other. There were six replicates in each compartment arranged in a split-plot randomized block design; inoculation treatments forming the main plots and cultivars the sub-plots. Assessments were carried out at three dates using a standard leaf area assessment key for leaf blotch. Account was also taken of the extent of necrosis not showing leaf blotch symptoms. When the plants were ripe, tiller number, ear number and grain weight were recorded; 1000 grain weight was calculated.

Results.

Plants inoculated with Race U.K.1 showed slower development than those inoculated with U.K.2, average growth stages being 44 and 52 respectively, at the first assessment (Table 48).

Table 48. Percentage leaf area, averaged over four top leaves, showing symptoms of Rhynchosporium secalis infection(R) and necrosis(N) in relation to inoculation treatment (transformed values).

Date	Growth Stage	early		early/late		late		control		SED±(DF=15)	
		R	N	R	N	R	N	R	N	R	N
		Race U.K.1									
14 June	44	4	<1	5	<1	0	0	0	0	2	<1
5 July	71	9	10	9	6	<1	0	<1	<1	2	3
18 July	86	10	23	9	22	2	20	2	17	1	5
		Race U.K.2									
14 June	52	21	5	21	5	0	0	0	0	3	2
5 July	78	20	38	19	46	3	5	4	8	2	3
18 July	93	5	66	5	70	5	79	3	78	2	11

At the first assessment (Table 48) plants inoculated with Race U.K.2 showed higher levels of infection than those inoculated with Race U.K.1. This difference continued at the second assessment when plants were at the milk stage of grain development. Late inoculation did not appear to add to infection levels, although there was some contamination of control plants at the second and third assessments. A high level of leaf necrosis was evident on plants inoculated with U.K.2 from 5 July and at the final assessment all plants inoculated with Race U.K.2 were in a state of senescence, irrespective of inoculation treatment. Rates of senescence of plants inoculated with Race U.K.1 were slower, associated partly with lower infection rates.

With respect to the effect of infection on components of yield (Table 49), there was no significant response associated in inoculation treatment and Race U.K.1.

Table 49. Effect of Rhynchosporium secalis inoculation treatment on components of yield.

Components of yield	Inoculation treatment				
	Early	Early/Late	Late	Control	SED±(DF=18)
Race U.K.1					
Tiller number	29	34	35	34	3
Ear number	18	24	20	26	3
Grain number	320	407	361	453	54
Grain weight(g)	13.5	16.8	16.2	20.2	2.5
1000 grain weight(g)	39.9	40.4	42.1	42.7	1.7
Race U.K.2					
Tiller number	41	40	40	42	3
Ear number	23	23	25	27	2
Grain number	326	310	403	409	47
Grain weight(g)	10.0	9.0	14.5	14.8	1.5
1000 grain weight(g)	29.6	28.3	34.7	36.3	1.5

Plants inoculated with Race U.K.2, however, showed a significantly reduced grain weight yield when inoculated at the early stage. This was associated with grain size rather than ear number although there was a small reduction in grain number. The effect of late inoculation, after ear emergence, was not significant.

The response of cultivars to infection by Race U.K.1 and U.K.2 is given in Tables 50 and 51 respectively. With Race U.K.1 Armelle showed no infection at the first assessment date (Table 50), while on other

Table 50. Percentage leaf area showing *Rhynchosporium secalis* infection and necrosis in relation to cultivar and leaf position (transformed values) Race U.K.1.*

Cultivar	Growth stage	*Rhynchosporium secalis* infection					Necrosis				
		leaf number					leaf number				
		1	2	3	4	mean	1	2	3	4	mean
14 June 1979											
Armelle	45	0	0	0	0	0	0	0	0	0	0
Dram	47	0	7	9	9	6	0	0	0	0	0
Golden Promise	47	0	5	3	2	2	0	0	0	0	0
Maris Mink	44	0	5	9	2	4	0	0	0	1	<1
Minak	41	0	7	13	4	6	0	0	0	0	0
Proctor	40	0	4	12	3	5	0	0	0	0	0
Vada	46	2	13	16	4	9	0	0	0	4	1
Mean	44	<1	6	9	3	5	0	0	0	1	<1
S.E.D.±(D.F.=120)	1	1	4	4	3	2	-	-	-	2	<1
5 July 1979											
Armelle	72	0	0	1	2	1	0	0	0	0	0
Dram	72	0	12	27	15	13	0	0	8	23	8
Golden Promise	74	0	8	18	8	8	0	3	9	25	9
Maris Mink	71	0	16	19	15	12	0	3	7	33	11
Minak	70	0	1	21	18	10	0	0	20	31	13
Proctor	64	0	1	15	16	8	0	0	7	18	6
Vada	74	1	9	21	12	11	0	5	11	16	8
Mean	71	<1	7	17	12	9	0	2	9	21	8
S.E.D.±(D.F.=120)	4	<1	3	4	3	2	-	2	5	8	3
18 July 1979											
Armelle	86	0	1	6	3	2	0	0	11	37	12
Dram	87	7	16	22	11	14	0	4	24	57	21
Golden Promise	89	8	10	9	7	8	13	25	49	79	41
Maris Mink	86	8	17	21	10	14	0	3	17	53	18
Minak	87	4	9	19	13	11	0	2	19	64	21
Proctor	79	3	2	15	15	9	0	0	17	46	16
Vada	87	5	12	15	3	9	0	7	36	69	28
Mean	86	5	10	15	9	10	2	6	25	58	22
S.E.D.±(D.F.=120)	3	3	3	5	4	2	4	7	13	14	7

* results derived from early inoculations.

Table 51. Percentage leaf area showing *Rhynchosporium secalis* infection and necrosis in relation to cultivar and leaf position (transformed values) Race U.K.2. *

Cultivar	Growth stage	*Rhynchosporium secalis* infection					Necrosis				
		leaf number					leaf number				
		1	2	3	4	mean	1	2	3	4	mean
14 June 1979											
Armelle	50	1	15	25	10	13	0	0	1	7	2
Dram	58	29	45	30	8	28	0	0	8	13	5
Golden Promise	51	15	23	21	8	17	0	2	10	8	5
Maris Mink	53	14	43	32	12	25	0	3	4	19	6
Minak	48	4	31	29	19	21	0	3	10	17	7
Proctor	49	0	11	30	28	17	0	0	4	5	2
Vada	52	18	33	35	25	28	0	2	9	18	7
Mean	52	12	29	29	16	21	0	1	7	12	5
S.E.D.±(D.F.=120)	2	6	8	8	8	4	-	2	5	8	3
5 July 1979											
Armelle	77	13	24	13	4	14	3	5	47	64	30
Dram	83	40	34	8	3	21	14	41	79	85	55
Golden Promise	81	29	28	10	0	17	4	30	62	83	45
Maris Mink	77	29	33	9	0	18	17	29	70	75	48
Minak	78	35	35	22	21	28	12	18	63	83	44
Proctor	73	17	24	31	14	22	3	15	53	73	36
Vada	79	23	27	8	2	15	3	21	52	68	36
Mean	78	27	29	14	6	19	8	23	61	76	42
S.E.D.±(D.F.=120)	3	6	6	5	4	3	4	8	13	12	7
18 July 1979											
Armelle	94	18	6	0	0	6	39	51	75	75	60
Dram	97	8	2	0	0	2	50	63	75	75	66
Golden Promise	97	2	2	0	0	1	68	70	75	75	72
Maris Mink	92	8	6	0	0	4	57	63	75	75	68
Minak	91	20	22	5	8	14	39	48	75	75	59
Proctor	88	14	12	0	0	6	49	62	83	83	69
Vada	94	4	0	0	0	1	85	83	83	83	83
Mean	93	11	7	1	1	5	55	63	77	77	68
S.E.D.±(D.F.=120)	2	5	4	3	4	3	12	10	5	5	6

* results derived from early inoculations.

cultivars infection was slight overall, being least on Golden Promise and most on Vada. Levels of infection at the second assessment were somewhat higher. At this time, Armelle showed very slight infection; Golden Promise and Proctor showing slightly less disease than the other remaining cultivars. These observations were repeated at the final assessment. All cultivars were susceptible to infection when inoculated with Race U.K.2 (Table 51). At the first assessment date Armelle showed less disease than other cultivars; Golden Promise and Proctor were intermediate. By the second assessment, considerable necrosis was associated with infection and there were no marked differences between cultivars, although Armelle tended to show fewer symptoms and Golden Promise and Proctor tended to be less infected on the flag leaf than other cultivars. At the final assessment necrosis was very high; Armelle, however, showed less leaf damage than other cultivars.

As already indicated, none of the inoculation treatments with Race U.K.1 had a significant effect on yield and there was no interaction between inoculation treatment and cultivar; although cultivars differed significantly with respect to tiller number, ear number, grain number, grain weight and 1000 grain weight. Thus variation could not be related to the resistance characteristics of the cultivars (Table 52).

Table 52. Yields (per pot) of cultivars inoculated with Race U.K.1 (means of all inoculation treatments).

Cultivar	tiller number	ear number	grain number	grain weight (g)	1000 grain weight(g)
Armelle	29	21	370	17	43
Dram	34	23	389	19	47
Golden Promise	26	17	325	11	34
Maris Mink	39	28	449	19	41
Minak	33	22	419	17	38
Proctor	36	16	286	11	37
Vada	35	26	460	22	47
S.E.D.±(D.F.=120)	2.3	2.5	50	2.3	1.7

Inoculation with Race U.K.2 was found to give significant yield reduction averaged over all cultivars (Table 49) but there was no significant interaction between inoculation treatment and cultivar and no evidence of a differential yield response to infection among cultivars (Table 53).

Table 53. Yields (per pot) of cultivars inoculated with Race U.K.2 (means of all inoculation treatments).

Cultivar	tiller number	ear number	grain number	grain weight (g)	1000 grain weight(g)
Armelle	38	21	354	14	38
Dram	43	28	407	16	38
Golden Promise	37	23	340	9	27
Maris Mink	45	27	360	11	28
Minak	47	29	412	14	32
Proctor	41	20	309	11	33
Vada	36	24	351	11	30
S.E.D.±(D.F.=120)	3.3	2.2	45	1.8	1.8

Thus, although Armelle showed less infection it did not appear to show a smaller yield reduction from infection resulting from early inoculation than other cultivars (Table 54).

Table 54. Percentage reduction in yield components from early inoculation with Rhynchosporium secalis for all cultivars, relative to late inoculation or uninoculated controls.

Cultivar	Race U.K.1			Race U.K.2		
	Grain number	Grain weight	1000 grain weight	Grain number	Grain weight	1000 grain weight
Armelle	4	4	7	18	36	22
Dram	+6	+1	3	+1	14	13
Golden Promise	+7	+3	1	9	30	20
Maris Mink	14	19	5	4[6]	57	19
Minak	36	43	11	19	33	18
Proctor	+13	+14	+5	24	32	11
Vada	23	34	13	27	46	26

DISCUSSION.

Studies with *Rhynchosporium secalis* were of a limited nature but do illustrate certain essential differences in the characteristics of the two diseases mildew and leaf blotch to be considered.

In early work difficulties were encountered in establishing conditions for successful infection by *R. secalis* but it was found that the use of an automatic misting system provided wet enough conditions to promote the progressive development of leaf blotch disease on the host plants. With this system differences in levels of resistance were found in lines from the European Disease Nursery and between commercial cultivars. Interactions between cultivar and race were indicated. Yield effects of *R. secalis* infection were also assessed and indicated that differences in yield response to *R. secalis* infection appeared to be less for different cultivars than with mildew. This may, however, relate to some degree to a lack of sensitivity to small differences which may have been masked in this experiment. It is also possible that the maintenance of a high himidity ameliorated the disease effects. Recent work (Habeshaw and Lennard, unpublished) shows that the major effect of *R. secalis* infection before the development of visible lesionsis on transpiration loss of water. The effect on photosynthetic rate is less where detached leaves receive sufficient water and the actual rate of photosynthesis during this period is influenced by the humidity of the surrounding air. It is also possible that an early threshold level is reached with leaf blotch disease beyond which further physiological damage ceases to affect the final yield. With pathogens such as *R. secalis* which act at a distance from the site of colonisation there is a less direct relation-

ship between the loss of CO_2 uptake during photosynthesis and the area of colonisation of the leaf (Habeshaw, 1979).

In leaf segment studies with *R. secalis* it was observed that lesion development and spore production level were not necessarily related; cultivars showing severe disease symptoms not always giving a high spore production rate. Habgood (1979) showed that although the final extent of infection and rate of sporulation of *R. secalis* varied with cultivar, size of lesions and time of lesion appearance did not. In keeping with the findings of this work older leaf tissue was found to be more resistant than younger.

Leaf segment studies did not show a close relationship with the findings from field observations on cultivar performance (N.I.A.B., 1980). Spore deposition in the field is however, more uncertain than in the laboratory and it is likely that in the field plants with an early prostrate growth habit may be more liable to infection.

GENERAL DISCUSSION

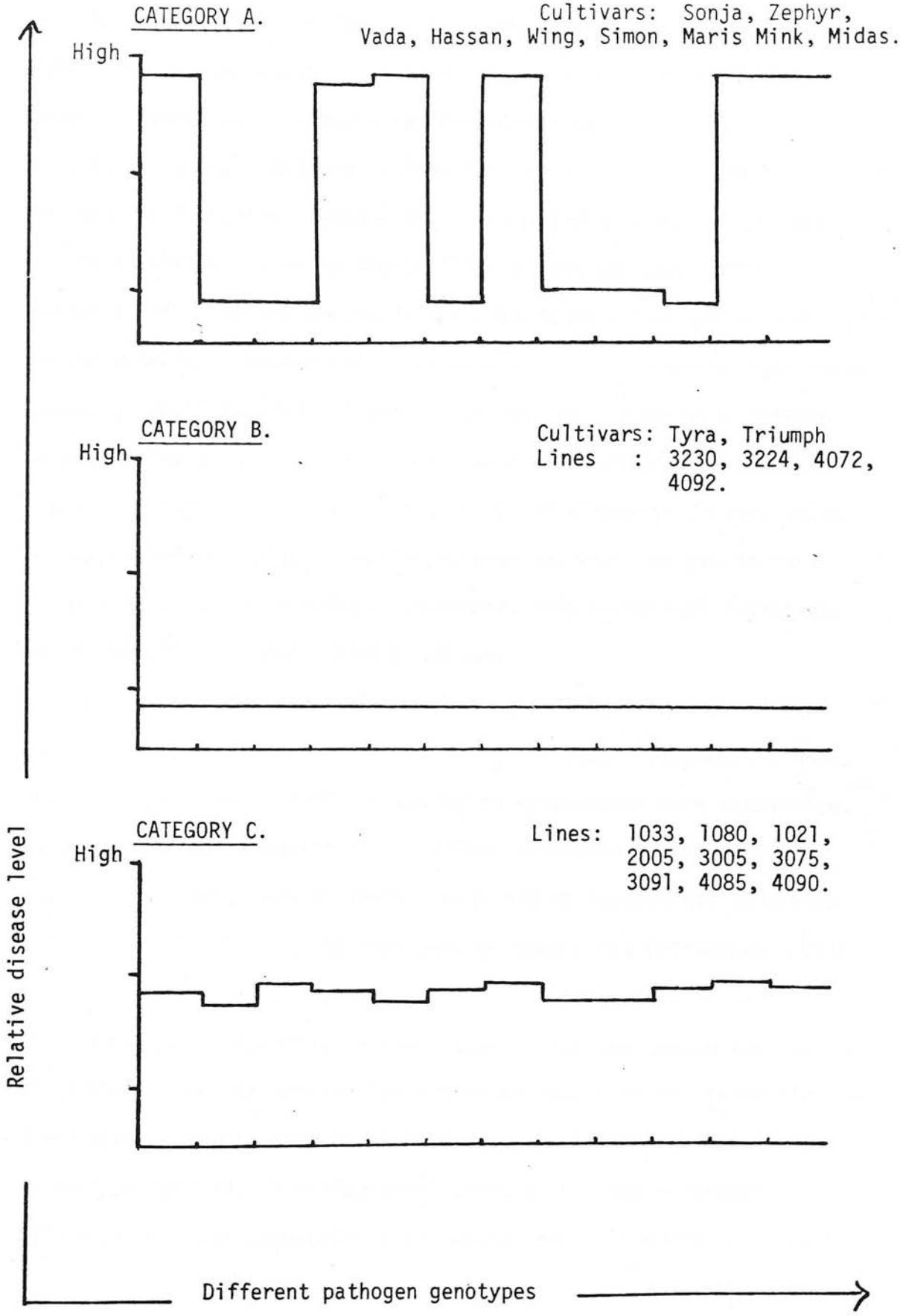
General Discussion.

The aim of this study was to explore some features of the plant-host/fungal-pathogen relationship and to consider some of the practical implications of different patterns in host response to infection by the pathogen. Two foliar diseases of barley were studied; mildew, which is of widespread economic importance and leaf blotch, a more sporadic and localised problem. Most of the work carried out related to mildew.

Preliminary screening tests of lines and cultivars from different barley collections (Experimental Section 1) revealed a wide range of responses to mildew infection. Commercial cultivars with known resistance factors, included in these investigations, were all infected to some degree in field and glasshouse trials,indicating the wide range of virulences present in the natural pathogen populations. The variation in responses of different lines included levels of susceptibility greater than those shown by any present day commercial cultivar. Some lines, however, showed consistently high and others consistently intermediate levels of resistance. Not all cultivars showed regular responses but those which did show consistently low levels of infection throughout were investigated further in a continuous cropping glasshouse experiment. Lines or cultivars which were uninfected in any trials or showed less than 1% infection on average were considered to be showing vertical resistance and were not studied further in this work. Alongside those lines selected for further study, cultivars with known resistance factors were grown. These became infected at the first growth cycle indicating the presence of virulence factors to overcome at least BMR factors 1-7. The universally susceptible cultivar Golden Promise was also grown and became heavily infected. In contrast, the selected lines showed a varying response, some eventually

proving susceptible, some maintaining a high level of resistance. In general, mildew levels increased with repeating cycles, perhaps indicating that the pathogen genotypes were becoming adapted to the host population. Leaf segment tests, inoculating consistently resistant lines and some commercial cultivars with isolates of *E. graminis* with different virulence factors, indicated three main categories of response in addition to that shown by the susceptible Golden Promise (Figure 16). Category A included most commercial cultivars which showed a characteristically vertical resistance pattern (Van der Plank, 1963). Triumph was difficult to categorize in this scheme and Tyra did not show a high level of infection with any isolate, failing to demonstrate the presence of virulence 7 in Race 18. Category B reflects a high level of resistance response to all isolates used, possibly due to the absence of corresponding virulence factors in the pathogen range explored. The range covered virulences apparently able to overcome one or other of the resistance groups present in the majority of British commercial cultivars. The lines 3230, 3224, 4072 and 4092 in this category may have major resistance genes not in commercial use in Britain. However, Tyra, with Mla resistance, was also in this catgeory, and this resistance may possibly have accounted for the response of the lines. Category C is perhaps of most interest as a potential source of durable resistance in as much as it reflects moderate disease levels with a wide range of virulences tested. Lines 1033, 1080, 1021, 2005, 3055, 3075, 3091, 4085 and 4090 in this category conform to the pattern of horizontal resistance (Van der Plank, 1963) and would be of interest for further study. Generally, in the reaction of cultivars to different *E. graminis* isolates, colony development did not always relate directly to the theoretical result expected after a consideration of the

Figure 16. Patterns of infection responses in lines and cultivars to different pathogen genotypes.



respective genotypes of host and named isolate of the pathogen, but may have partly been due to some contamination.

Studies in Experimental Section 2, with a range of commercial cultivars grouped according to their reported resistance factors, showed differences in disease ratings associated with major gene effects. However, disease ratings for cultivar groups altered between the two years of the experiment, presumably due to changes in the virulence genotypes present in the pathogen population. Variation of a smaller magnitude was also found within groups and could be attributable to background resistance genes. The differences were, however, generally small, inconsistent, and not necessarily durable. As previously discussed, Wolfe and Schwarzbach (1979), for example, reported changes in U.K. infection levels of mildew on Zephyr, Julia and Deba Abed, all possessing Mlg resistance, with changes in their respective areas of cropping. Moreover, this background resistance may be modified by environmental factors.

Evidence of background resistance persisting was obtained in studies on old cultivars with no known major gene resistance. Asse, described by Russell (1978) as having no known major gene resistance, showed considerable resistance to mildew infection in the present studies. Others, such as Ymer, proved highly susceptible to mildew infection although they had been popular when first introduced. This could have been due to a lower intensity of barley growing at the time and a reduced availability of host material for the growth and proliferation of mildew. Despite the similar infection levels which ultimately developed on Proctor and Golden Promise, variations did occur in the respective patterns of mildew development on the two cultivars. Tolerance has been associated with Proctor and this might be related

to delayed infection development indicated by the comparatively low mildew ratings for the first leaf. The rate of infection of the newly emerging leaf has received little attention but it would be of interest to explore the physiological implications of the variation in the infection patterns on developing leaves. A further feature of Proctor was a tendency to show less necrosis in response to infection than Golden Promise. Habeshaw (1979) has measured differences in the effects of infection on rates of photosynthetic Co_2 uptake in different cultivar/pathogen combinations. In "compatible" responses, associated with relatively little necrosis, there was less of a reduction in photosynthetic activity on the leaf as a whole, than where a great amount of necrosis "at a distance" from the site of colonization occurred.

In assessing the responses of cultivars with Mlg resistance, there appeared to be fewer cultivars with good background resistance relative to cultivars with no major gene resistance. For example, three out of 15 cultivars with no major gene resistance showed an overall mean infection level of less than 5 per cent in two glasshouse experiments. In a parallel study, using 12 cultivars possessing Mlg resistance, none came into this low disease level category. However, in this group there were significant differences in background resistance levels and Armelle was often less infected than other cultivars. In some cases this reduced disease level was more evident at later growth stages. Such resistance, although at an intermediate level may still be advantageous in the field in slowing the rate of epidemic development during crop growth: it may also provide a yield advantage.

From studies on mildew development at the microscopic level

(Experimental Section 3) of various host/pathogen combinations it was indicated that most resistance responses were associated with cessation of fungal growth at the appressorial stage. Koga *et al* (1978), in studies on incompatible interactions in barley leaves inoculated with *E. graminis*, found that more than 80% of parasitic units ceased growth at the first stage of papilla formation: the more resistant the host the more conidia failed to establish. These results are in keeping with the present observations which indicated that even where isolates showed an absence of virulence factors to overcome resistance in a particular cultivar, a proportion of conidia still produced sporulating colonies. The results of the microscopic assessments of intermediate levels of resistance failed to identify factors which may have had a significant role in disease development. This may have been due to the choice of qualitative criteria: an assessment of quantitative factors such as the lengths of periods of latency and of infectiousness and the rate of spore production may have provided a better insight into factors involved. There was evidence, however, that a plant or leaf age factor affected fungal development. Fewer sites of infection showed development beyond the appressorial stage on leaves from plants at later growth stages, but older leaves from the lower leaf position on the plant up to the fourth leaf supported a more advanced fungal growth.

The final studies (Experimental Section 4) concerned the development of *Rhynchosporium secalis* on barley lines and cultivars, although initially some difficulties were encountered in initiating epidemics. Seedling experiments on the European and Expanded European Barley Disease Nurseries demonstrated the difficulties in the establishment of leaf blotch in comparison with mildew; use of automatic misting

systems overcame this, however. Experiments with two races U.K.1 and U.K.2 on commercial cultivars also gave variable results, where a manual misting system was applied, again demonstrating the great sensitivity of the pathogen to its environment.

With respect to physiological responses of the plant to *R. secalis* infection, disease development appears to be an expression of active enzyme/toxin activity as well as of fungal growth and development. The fungus can act "at a distance" and thus there is a less direct relationship between loss in photosynthetic activity and actual colony area than in the case of mildew on a compatible host (Habeshaw, 1979). Yield responses to *R. secalis* infection did not appear to follow the results of disease assessments of different cultivars when infection was substantial on all. This suggests that differences in amount of infection above a certain critical threshold may lead to equal impairment of yield. Moreover, the effects of leaf blotch on the plant as a whole may be governed to a much greater extent by its position on the leaf than is the case with mildew. Leaf blotch infection disrupts the underlying leaf tissue and a lesion towards the base of the leaf may affect the functioning of the entire leaf. This may also account for inconsistencies between disease assessments.

In studies on colony development it was observed that the size of the lesion did not necessarily correspond to the numbers of spores produced as would tend to be the case with mildew. Assessment of disease severity may not therefore, reflect epidemic potential. This may be of more significance in a season of low infection levels than when levels are high, if there is a threshold value of infection above which crop loss does not increase with infection.

APPENDICES

APPENDIX I. Lines and cultivars assessed in this study (*marks those selected for further study).

Expanded European Barley Disease Nursery

Study number	Name
1001	Sultan
1002	C.I. 8251
1003	Ariana
1004	Arlington Awnless
1005	Chiro Chinko
1006	Gujrat
1007	Hor 80215
1008	Lyallpur B.S.
1009	" 3645
1010	Agio
1011	L.96
1012	L.100
1013	C.I. 4219
1014	Spiti
1015	M.C. 20
1016	Roger's Winter Barley
1017	57/510-44
1018	Hor 992
1019*	Hor 1036
1020	Hor 1630
1021*	Ab. 3 S3176/61
1022	Ab. 6 Hor 1677
1023	Ab.12 Hor 1718
1024	Ab.15 Hor 1643
1025	Ab.16 Hor 1537
1026	Ab.23 Hor 1610
1027	Ab. 116/47
1028	E.P. 71
1029	E.P. 74
1030	E.P. 76
1031	Medium 026
1032	Hor 1457
1033*	L.97
1034	Ab. 6208/48
1035	Grannelose 2 Zeilige
1036	Lyallpur P
1037	Vierzeilige BAA 810
1038	Proctor
1039	Nigrisubnudum
1040	Ianthinum H.E.S. 162
1041	I.5
1042	I.25
1043	C.I. 9588
1044	No.6018
1045	Hor 1884
1046	H. 2212
1047	E. 1388
1048	F. 784
1049	G. 1028
1050	I. 265

Study number	Name
1051	P. 1647
1052	I. 2146
1053	Mianwali
1054	A. 222
1055	Palmella Blue
1056	Russian 12
1057*	Vagabond
1058	Linie 4831
1059	Lyallpur 3647
1060	Svalöv 64505
1061	Svalöv 65505
1062	Maryland
1063	Quinn
1064	Gold
1065	Bolivia
1066	Estate
1067	Rika # 1
1068	Cebada Capa
1069	Ricardo
1070	Lechtaler
1071	Sudan
1072	Speciale
1073	Aim
1074	Berg
1075	Belfor
1076	Julia
1077	C.I. 10241
1078	C.I. 12126
1079	Astakus
1080*	C.I. 13988
1081	Clermont
1082	Mutant R.F.7
1083	" A.3502
1084	" 7085
1085	B.294
1086	C.I. 4974
1087	San Carlos
1088	C.I. 12201
1089	C.I. 12202
1090	C.I. 12203
1091	X 7
1092	X 21
1093	Hor 1036
1094	Voldagsen 8141
1095	C.I. 2376
1096	Arabische
1097	Heine 4808
1098	Cambrinus
1099	Ethiopian 1
1100	Gun Eki

APPENDIX I. (cotinued).

Expanded European Barley Disease Nursery.

Study number	Name
1101*	Marco
1102	Oliveros 1
1103	" 3
1104	Australische No.22
1105	Maris Canon
1106	Algerian
1107	Goldfoil
1108	Kwan
1109	Psaknon
1110	Multan
1111	Glumes
1112	Rupee

Study number	Name
1113	Minerva
1114	C.I. 1243
1115	E.P.72=L137
1116	Zweizeilige BAA 822
1117	Djob A
1118	Hor 728
1119	Weider
1120	Marret Puntress
1121	Aramir
1122	Pirouette
1123	Valeta

European Barley Disease Nursery.

Study number	Name
2001	Abyssinian 14
2002	" 26
2003	" 1102=L94
2004	" 1128=L98
2005*	" 1139=L199
2006	X 33
2007	Bey
2008	Bigo
2009	Cerès
2010	Golden Promise
2011	C.I. 1237
2012	C.I. 1243
2013	Debat
2014	Decorticatum
2015*	E.P.73
2016	E.P.75
2017*	E.P.77
2018	E.P.79=L92
2019	Emir
2020	Golden Promise
2021	Engledow India
2022	La Estanzuela
2023	Freja x Jerusalem
2024	Gondar
2025	Dans Irisaka 8-31

Study number	Name
2026	Japan 1
2027	Jerusalem II
2028	Lyallpur 3647
2029	Menelik
2030	Golden Promise
2031	Monte Cristo
2032	Nigrinudum
2033	Russian 81
2034	X 27
2035	Hor 1063
2036	Hor 1402
2037	Nigrate
2038	Abed Binder 12
2039	I 5
2040	Golden Promise
2041	Volla
2042	Wisconsin H42
2043	Batna
2044	No.5678
2045	No.7372
2046	Minerva
2047	Zweizeilige BBA 822
2048	Hor 728
2049	Nepal 81
2050	Golden Promise
2051	C.I. 8503

APPENDIX I. (continued)

Ethiopian Collection

Study number	Name	Species/source
3001	382666	UM N.E. ADDIS ABA
3002*	" 669	RE "
3003	" 671	UM "
3004*	" 676	RE "
3005	" 677	UM "
3006	" 679	RE "
3007	" 680	RE "
3008*	" 683	UM "
3009*	" 684	RE "
3010	" 685	RE "
3011	" 687	RE "
3012	" 689	UM "
3013*	" 691	RE "
3014	" 693	UM "
3015*	" 694	RE "
3016	" 696	UM N. ADDIS ABA
3017	" 698	UM "
3018*	" 700	UM "
3019*	" 702	RE "
3020	" 704	UM "
3021	" 706	RE "
3022	" 708	UM "
3023	" 711	RE "
3024*	" 712	RE "
3025	" 716	RE "
3026	" 718	UM "
3027	" 720	UM "
3028	" 722	RE "
3029	" 724	UM "
3030	" 731	UM S. Gondar
3031	" 732	UM "
3032	" 735	UM "
3033	" 738	UM "
3034	" 740	RE "
3035	" 743	UM "
3036	" 746	UM "
3037	" 752	RE "
3038	" 753	RE N. Gondar
3039	" 755	UM "
3040	" 759	UM "
3041	" 765	UM "
3042	" 766	UM "
3043 *	" 771	UM "
3044*	" 773	RE "
3045	" 788	RE "
3046	" 790	UM "
3047	" 798	RE "
3048	" 702	UM "
3049	" 714	RE "
3050	" 817	UM "

Study number	Name	Species/source
3051	382818	RE N. Gondar
3052*	" 819	UM "
3053	" 821	RE "
3054	" 826	RE "
3055*	" 828	UM "
3056	" 830	RE "
3057	" 834	UM "
3058	" 847	UM "
3059	" 868	RE "
3060	" 874	RE "
3061	" 878	UM "
3062*	" 885	RE W. Axam
3063	" 886	RE "
3064	" 890	UM "
3065*	" 891	RE "
3066*	" 892	RE N. Adowa
3067*	" 895	UM E. Intcho
3068	" 897	RE "
3069	" 901	RE "
3070	" 907	UM "
3071	" 908	RE "
3072	" 915	RE N.E. Intcho
3073	" 916	UM "
3074	" 924	RE S. Adigrat
3075*	" 927	RE "
3076	" 930	RE "
3077	" 932	RE "
3078	" 836	UM S. Mekele
3079	" 940	RE "
3080	" 945	RE "
3081	" 948	UM "
3082	" 951	RE "
3083	" 961	RE "
3084	" 962	UM "
3085	" 972	RE "
3086*	" 976	UM "
3087	" 984	RE "
3088	" 988	RE "
3089	" 993	RE "
3090*	" 997	UM "
3091*	383005	RE "
3092	" 006	UM S. Kombokha
3093	" 067	RE "
3094	" 017	RE "
3095	" 018	RE "
3096	" 020	UM SW ADDIS ABA
3097	" 021	UM "
3098*	" 023	RE N.E. Jimma
3099	" 024	RE W. AMBO
3100	" 026	UM "

APPENDIX I. (continued)

Ethiopian Collection.

Study number	Name	Species/source
3101	383028	RE W. AMBO
3102	" 034	RE "
3103*	" 036	RE "
3104	" 037	RE "
3105	" 038	RE "
3106	" 039	RE "
3107	" 041	UM "
3108	" 042	RE "
3109	" 048	UM "
3110	" 052	RE "
3111	" 057	UM "
3112*	" 059	UM "
3113	" 065	RE N.W. AMBO
3114	" 074	UM "
3115*	" 077	RE "
3116	" 085	UM "
3117	" 087	RE "
3118	" 089	UM "
3119	" 092	UM "
3120	" 094	RE "
3121	" 098	UM "
3122	" 101	RE Fincha
3123	" 103	UM "
3124*	" 106	RE "
3125	" 111	UM E. AMBO
3126*	" 116	UM "
3127	" 119	UM "
3128	" 120	RE "
3129	" 124	RE "
3130	" 126	UM "
3131	" 130	UM S.E. AA
3132	" 132	UM "
3133	" 133	UM "
3134	" 135	UM "
3135	" 140	RE "
3136	" 144	UM "
3137	" 146	UM "
3138	" 151	RE "
3139	" 156	RE "
3140	" 158	UM "
3141	" 167	RE "
3142*	" 175	RE "
3143	" 176	UM "
3144	" 179	RE "
3145	" 184	RE "
3146	" 188	RE "
3147	" 193	UM "
3148*	" 194	UM "
3149	" 199	RE "
3150	382184	NS NS N.ADDIS ABA

Study number	Name	Species/source
3151	382187	NS N.ADDIS ABA
3152	" 188	NS N.BHAR DAR
3153	" 190	NS "
3154	" 194	NS N.Gondar
3155	" 197	NS "
3156	" 201	NS "
3157	" 203	NS "
3158	" 225	NS W. Axum
3159	" 228	NS "
3160	" 231	NS "
3161	" 235	NS "
3162	" 239	NS E. Axum
3163	" 240	NS "
3164	" 246	NS "
3165	" 248	NS N. Adowa
3166	" 252	NS E. Axum
3167	" 253	NS "
3168	" 260	NE E. Adowa
3169	" 266	NS "
3170	" 269	NS Intcho
3171	" 273	NS N.E.Intcho
3172	" 282	NS "
3173	" 290	NS E. Intcho
3174	" 294	NS S. Adigrat
3175	" 302	NS "
3176	" 304	NS "
3177	" 308	NS "
3178	" 309	NS "
3179	" 311	NS "
3180	" 313	NS "
3181	" 315	NS S. Mekele
3182*	" 317	NS "
3183	" 326	NS "
3184	" 331	NS "
3185	" 333	NS "
3186	" 342	RE "
3187	" 344	RE "
3188	" 345	NS "
3189	" 353	NS "
3190	" 358	NS "
3191	" 361	NS W. ADDIS ABA
3192*	" 372	NS N.E. Jimma
3193	" 373	NS S.W.ADDIS ABA
3194	" 376	NS W. AMBO
3195	" 391	NS "
3196	" 410	NS N.W. AMBO
3197	" 413	NS Fincha
3198	" 418	NS S.E. AA
3199	" 424	NS
3200	" 429	NS

APPENDIX I. (continued).

Ethiopian Collection.

Study number	Name	Species/source
3201	382432	NS
3202	" 435	NS
3203	" 438	NS
3204	" 441	NS
3205	" 447	NS
3206	" 448	NS
3207	" 452	NS
3208	" 457	NS
3209	" 461	ON W. Axum
3210*	" 466	ON E. Axum
3211	" 467	ON N. Adowa
3212	" 471	ON "
3213	" 477	ON E. "
3214	" 477	ON "
3215	" 479	ON Intcho
3216	" 481	ON N.E. Intcho
3217	" 488	ON "
3218	" 490	ON "
3219	" 491	ON E. Intcho
3220	" 495	ON "
3221	" 509	ON S.A. Digat
3222	" 511	ON S.W. AA
3223	" 513	ON "
3224*	" 514	ON N.E. Jimma
3225*	" 517	ON "

Study number	Name	Species/course
3226	382532	ON N.E. Jimma
3227	" 571	ON "
3228	" 575	ON S.W. AA
3229	" 581	ON "
3230*	" 585	ON W. AMBO
3231	" 588	ON "
3232	" 598	ON "
3233	" 602	ON "
3234	" 603	ON "
3235	" 605	ON "
3236	" 607	ON "
3237	" 611	ON "
3238	" 613	ON N.W. AMBO
3239	" 615	ON E. AMBO
3240	" 620	OH Holetta
3241	" 641	E. N.A.A.
3242*	" 650	E. N.C. Jimma
3243	" 655	E. W. AMBO
3244	" 663	E.E. AMBO

Hordeum Spontaneum Collection.

Study number	Name
4001	282527
2	" 575
3	" 577
4	" 583
5	" 586
6	" 587
7	" 600
8	" 608
9	" 609
4010	" 613
1	" 616
2	" 620
3	" 621
4	" 631
5	" 636
6	" 637
7	" 638
8	" 640
9	" 642
4020	" 644

Study number	Name
4021	282645
4022	" 646
4023	" 660
4024	" 661
4025	" 636
4026	" 665
4027	" 666
4028	" 669
4029	" 670
4030	" 672
4031	" 674
4032	" 677
4033*	" 679
4034*	" 683
4035	284742
4036	" 743
4037	" 749
4038	" 750
4039	" 753
4040	" 754

Study number	Name
4041	284755
4042	296796
4043	" 800
4044	" 801
4045	" 802
4046	" 803
4047	" 812
4048	" 813
4049	" 815
4050	" 821
4051*	" 827
4052	" 829
4053	" 830
4054*	" 831
4055	" 834
4056	" 835
4057	" 836
4058	" 838
4059	" 839
4060	" 850

APPENDIX I. (continued).

Hordeum Spontaneum Collection.

Study No.	Name
4061	296853
4062	" 854
4063	" 856
4064	" 861
4065	" 863
4066*	" 864
4067	" 865
4068	" 873
4069	" 874
4070	" 875
4071	" 877
4072*	" 878
4073	" 881
4074	" 882
4075	" 884
4076	" 885
4077	" 886
4078	" 892
4079	" 899
4080	" 904
4081	" 912
4082	" 913
4083	" 915
4084*	" 919
4085*	" 920
4086	" 921
4087	" 922
4088	" 932
4089	" 933
4090*	" 934
4091*	" 935
4092*	" 942
4093*	" 944
4094*	" 945
4095	" 952
4096	" 956
4097	282599
4098	" 611
4099	" 591
4100	" 635
4101*	349803
4104	" 808
4105	" 809
4108	354926
4109	" 927
4110	" 928
4111	" 929
4112	" 936
4113	" 937
4114*	" 938
4115	" 939
4116	354942
4117	" 942
4118	" 947
4119	" 949

Turkish Collection.

Study No.	Name
5001	245740
5002	245742
5003	342014
5004	" 017
5005	" 018
5006*	" 019
5007	" 021
5008	" 022
5009	" 023
5010*	" 025
5011	" 026
5012	" 029
5013	" 030
5014	" 031
5015	" 032
5016	" 033
5017	" 034
5018	" 035
5019	" 035
5020	" 037
5021	" 038
5022	" 039
5023*	" 040
5024	" 041
5025	" 042
5026	" 043
5027	" 044
5028	" 045
5029	" 046
5030	" 047
5031	" 048
5032	" 049
5033	" 050
5034	" 052
5035*	" 053
5036	" 055
5037	" 056
5038	" 057
5039	" 058
5040	" 059
5041	" 060
5042	" 061
5043	" 062
5044	" 063
5045	" 064
5046	" 065
5047	" 066
5048	342068
5049	" 069
5050	" 070
5051	" 071
5052	" 072
5053	" 076
5054	" 077
5055	" 078
5056	" 079
5057	" 080
5058	" 081
5059	" 082
5060	" 083
5061	" 084
5062	" 085
5063	" 086
5064	" 087
5065	" 088
5066	" 089
5067	" 090
5068	" 091
5069	" 092
5070	" 093
5071	" 094
5072	" 095
5073	" 098
5074	" 099
5075	" 100
5076	" 101
5077	" 102
5078	" 103
5079	" 104
5080	" 105
5081	" 106
5082	" 107
5083	" 108
5084	" 109
5085	" 110
5086	" 111
5087	" 112
5088	" 114
5089	" 116
5090	" 117
5091	" 118
5092	" 119
5093	" 120
5094	" 121
5095	" 122
5096	" 123
5097	" 124
5098	" 125
5099	" 126
5100	" 127

APPENDIX I. (continued)

Turkish Collection

Study No.	Name
5101	342128
5102	" 129
5103	" 130
5104	" 131
5105	" 132
5106	" 133
5107	" 134
5108	" 135
5109	" 136
1110	" 137
5111	" 138
5112	" 139
5113	" 140
5114	" 141
5115	" 142
5116	" 143
5117	" 144
5118	" 145
5119	" 146
5120	" 147
5121	" 148
5122	" 149
5123	" 150
5124	" 151
5125	" 152
5126	" 153
5127	" 154
5128	" 155
5129	" 156
5130	" 157
5131	" 158
5132	" 159
5133	" 160
5134	" 161
5135*	" 162
5136	" 163
5137	" 164
5138	" 165
5139	" 166
5140	" 167
5141	" 168
5142	" 169
5143	" 170
5143	" 171
5145	" 172
5146	" 173
5147	" 174
5148	" 175
5149	" 176
5150	" 177

Study No	Name
5151	342178
5152	" 179
5153*	" 180
5154	" 181
5155	" 182
5156	" 183
5157	" 184
5158	" 185
5159	" 186
5160	" 187
5161	" 188
5162	" 189
5163	" 190
5164	" 191
5165	" 192
5166	" 193
5167	" 194
5168	" 195
5169	" 196
5170	" 197
5171	" 198
5172	" 199
5173	" 200
5174	" 201
5175	" 202
5176	" 203
5177	" 205
5178	" 206
5179	" 207
5180	" 208
5181	" 209
5182	" 210
5183	" 212
5184	" 213
5185	" 215
5186	" 217
5187	" 218
5188	" 219
5189	" 220
5190	" 221
5191	" 222
5192	" 225
5193	" 226
5194	" 227
5195	" 228
5196	" 230
5197	" 231
5198	" 232
5199	" 233
5200	" 235

Study No.	Name
5201	342236
5202	" 238
5203	" 240
5204	" 241
5205	" 24=
5206	" 243
5207	" 245
5208	" 246
5209	" 247
5210	" 248
5211	" 249
5212	" 250
5213	" 251
5214	" 252
5215	" 253
5216	" 254
5217	" 255
5218	" 256
5219	" 257
5220	" 258
5221	" 259
5222	" 260
5223	" 261
5224	" 262
5225	" 263
5226	" 264
5227	" 265
5228	" 266
5229	" 267
5230	" 268
5231	" 269
5232	" 270
5233	" 271
5234	" 273
5235	" 274
5236	" 275
5237	" 276
5238	" 277
5239	" 278
5240	" 279
5241	" 280
5242	" 281
5243	" 282
5244	" 283
5245	" 284
5246	" 285
5247	" 286

APPENDIX I. (continued).

Commercial Collection.

Study No.	Name
6001	C.P. 127422
6002	Goldfoil
6003	Ragusa b
6004	W 27/136
6005	W 14/145
6006	Rinn 4
6007	Maris Concorde
6008	M511
6009	M515
6010	H501
6011	H1063
6012	H1657
6013	H1036
6014	Nigerian
6015	H1104
6016	H1402
6017	Akka
6018	Iris
6019	Sultan
6020	Tein
6021	Vada
6022	Proctor
6023	Wing
6024	Weibulls 1001
6025	MC20
6026	Ming
6027	Fong Tein
6028	Aramir
6029	Emir
6030	Abacus
6031	Berac
6032*	Hassan
6033	Imber
6034	Julia
6035	Lofa Abed
6036	RPB 57/69
6037	1817xRPB 257/67
6038*	Midas
6039	Universe
6040*	Golden Promise
6041*	Mazurka
6042*	Wing
6043	Sultan
6044*	Zephyr
6045*	Vada

Study No.	Name
6046*	Maris Mink
6047	Ymer
6048	Maythorpe
6049	Proctor
6050	Porthos
6051	Astrix
6052	Athos
6053	Maris Otter
6054*	Tyra
6055	Sundance
6056	Gorgie
6057	Lami
6058	Varunda
6059	Armelle
6060	Gerka
6061	Ark Royal
6062*	Sonja
6063	Asse
6064	Nymphe
6065	1817xNymphe
6066	1817xAsse
6067	Tern
6068	♂→ sterile
6069	Bigo
6070	Cambrinus
6071	Plumage Archer
6072	Gull
6073	Deba Abed
6074	Yamina
6075	Luke
6076	Dram
6077	Koru
6078	Athene
6079	Katy
6080	Mala Abed

APPENDIX II. Percentage mildew averaged over top four leaves of those lines selected for further assessment (G.S.40).

Line number	Field	Unheated Glasshouse	Heated Glasshouse	Line number	Field	Unheated Glasshouse	Heated Glasshouse
1019	-	-	5	3115	1	25	50
1021	1	5	5	3122	0	25	5
1033	1	5	5	3124	1	0	10
1057	5	0	5	3126	5	0	10
1080	1	0	5	3142	1	5	10
1101	0	10	5	3148	1	0	10
				3182	5	0	10
2005	5	10	5	3192	1	10	10
2015	5	0	5	3210	10	10	10
2017	5	10	5	3224	1	0	10
				3225	1	0	5
3002	1	0	5	3230	1	0	10
3003	5	5	15	3242	5	0	10
3004	-	0	10				
3008	5	0	15	4033	-	0	10
3009	-	0	15	4034	-	10	0
3013	0	0	10	4051	-	10	0
3015	1	0	10	4054	-	0	5
3018	10	0	15	4066	-	10	0
3024	1	0	10	4072	-	5	0
3043	1	0	5	4084	-	10	0
3044	10	0	5	4085	-	10	0
3019	5	0	25	4090	-	10	0
3052	5	-	10	4091	-	10	0
3055	1	0	15	4092	-	10	0
3062	5	0	5	4093	-	10	0
3065	1	0	15	4094	-	5	0
3066	1	10	15	4101	-	10	5
3067	5	10	15	4114	-	10	10
3075	1	0	5				
3086	5	10	10	5006	10	0	10
3090	-	10	10	5010	10	0	10
3091	10	10	10	5023	5	10	10
3098	5	0	10	5135	5	0	10
3103	1	10	10	5153	5	10	10

APPENDIX III. Percentage mildew (transformed values) averaged over four upper leaves for lines and cultivars grown over seven cycles.

Table IIIa. Cycles 1-4.

line/ cultivar number	Cycle number									
	1				2			3		4
	Growth stage									
	32	33	40	51	22	28	41	22	31	36
1019	0	0	0	0	2	0	0	0	0	0
1021	1	0	0	2	0	0	0	0	0	0
1033	2	0	0	0	0	0	0	0	0	0
1057	0	0	0	4	1	4	2	3	6	0
1080	2	1	1	1	3	2	2	7	1	0
1101	0	2	0	0	6	0	0	10	3	0
2005	0	0	0	0	0	1	1	0	0	0
2015	4	2	6	12	11	0	0	12	2	0
2017	0	2	6	0	4	0	0	1	3	0
3002	1	0	2	3	5	4	0	13	5	0
3004	4	1	1	0	4	8	3	10	2	0
3008	4	2	7	1	4	3	1	9	10	0
3009	0	0	0	6	1	2	0	12	4	0
3013	2	0	4	2	6	0	0	4	5	0
3015	1	0	1	4	1	6	5	10	0	0
3018	0	0	8	0	11	4	7	11	7	0
3024	2	0	0	0	9	2	3	20	3	0
3043	0	0	1	1	3	6	1	17	5	0
3044	0	0	1	0	3	3	6	13	6	0
3019	3	5	4	0	16	5	0	23	15	0
3052	0	0	1	5	6	5	2	14	2	0
3055	0	1	9	0	7	5	3	7	7	0
3062	0	0	0	0	1	0	1	10	4	0
3065	6	2	3	0	9	8	0	21	8	0
3066	4	2	0	4	22	11	3	10	3	0
3067	6	2	5	2	9	5	1	11	8	0
3075	0	0	0	6	2	0	0	1	1	0
3086	2	0	4	1	12	2	8	21	6	0
3090	1	0	5	4	1	3	0	10	5	0
3091	1	0	0	0	6	0	5	2	0	0
3098	1	4	8	6	8	1	0	13	2	0
3103	0	0	3	2	2	0	0	11	6	0
3115	0	0	2	2	15	0	3	7	10	0
3122	0	1	0	3	8	2	2	12	3	0
3124	2	0	3	2	1	0	3	11	3	0
3125	0	0	3	0	3	0	0	6	4	0
3126	0	0	2	0	9	1	0	6	2	0
3142	2	0	7	0	1	0	0	9	2	0
3148	0	2	0	0	4	0	0	11	1	0
3182	6	2	7	1	15	2	5	23	9	0
3192	0	1	2	0	11	6	0	10	4	0

Table IIIa:- continued.

line/ cultivar number	Cycle number									
	1				2			3		4
	Growth stage									
	32	33	40	51	22	28	41	22	31	36
3210	2	0	5	6	15	2	1	14	8	0
3224	0	0	2	1	11	0	3	4	5	0
3225	4	3	2	1	2	5	0	10	7	0
3230	0	0	0	1	12	5	5	8	9	0
3242	0	0	3	0	1	3	0	9	4	0
4033	0	0	0	0	0	0	0	10	5	0
4034	1	0	0	0	1	4	3	10	5	0
4051	0	0	0	0	1	0	0	10	5	0
4054	0	0	0	0	0	3	3	10	8	0
4066	0	0	8	0	0	1	0	1	1	0
4072	10	0	0	0	1	0	0	0	0	0
4084	0	0	0	0	2	0	0	0	0	0
4085	1	0	0	0	1	4	0	0	0	0
4090	1	0	0	0	0	0	0	0	0	0
4091	0	0	0	0	0	0	0	10	1	0
4092	0	0	0	0	2	1	0	3	0	0
4093	0	0	0	4	3	2	0	5	0	0
4094	0	0	0	0	0	0	0	0	0	0
4101	2	0	7	0	1	0	0	10	5	0
4114	0	2	0	0	0	4	0	4	1	0
5006	2	3	2	1	15	8	6	12	4	0
5010	6	2	9	0	21	13	11	12	8	0
5023	9	7	16	3	30	14	5	24	12	8
5135	2	1	1	1	10	2	1	9	0	0
5153	4	1	8	3	5	0	0	1	1	0
6032	3	2	6	6	21	7	0	21	12	0
6038	8	7	7	6	30	18	5	22	13	0
6040	9	5	5	6	33	21	10	27	17	0
6041	0	0	2	2	6	7	1	14	8	0
6042	0	0	2	7	9	8	10	23	9	0
6044	8	7	11	11	22	12	12	24	11	0
6045	3	0	6	2	21	1	3	17	14	0
6046	1	0	0	2	9	7	0	10	4	0
6054	0	0	0	0	5	8	8	9	6	0
6062	6	2	11	2	14	11	8	20	2	0
mean	2	1	3	2	7	4	2	10	5	1
S.E.D.±	1.8	1.7	2.9	2.7	4.2	3.6	3.1	4.5	3.4	0.1
(D.F.)	(212)	(220)	(150)	(223)	(158)	(202)	(218)	(193)	(190)	(72)

Table IIIb. Cycles 5-7.

line/ cultivar number	Cycle number								
	5			6				7	
	Growth stage								
	29	32	39	27	35	29	50	28	31
1019	3	2	2	7	7	8	6	8	8
1021	0	0	1	0	3	2	5	3	9
1033	0	0	0	4	2	2	4	3	3
1057	6	0	4	4	2	8	6	11	8
1080	3	1	0	0	2	2	0	9	7
1101	0	0	0	7	8	4	6	7	6
2005	0	0	0	0	2	2	1	0	1
2015	3	0	0	7	2	8	6	8	10
2017	0	2	1	6	5	5	6	8	12
3002	1	1	0	4	11	15	9	12	12
3004	1	2	1	5	1	2	6	8	6
3008	4	2	3	3	7	8	30	8	2
3009	4	2	1	8	3	4	6	6	8
3013	0	1		3	3	6	6	1	3
3015	0	3	8	10	5	3	4	2	7
3018	4	5	7	2	2	8	18	8	8
3024	8	0	3	9	8	4	17	2	8
3043	1	2	1	12	22	4	1	4	5
3044	0	4	1	8	2	1	2	4	16
3019	6	0	4	3	8	8	6	8	10
3052	1	8	0	9	12	7	6	9	7
3055	2	0	0	8	5	10	6	8	9
3062	3	0	1	0	3	11	6	8	10
3065	2	1	2	7	12	22	6	8	5
3066	8	2	2	15	7	15	6	8	8
3067	5	3	0	9	3	8	6	14	8
3075	2	0	0	2	2	8	2	3	2
3086	6	4	4	7	10	8	6	9	7
3090	4	3	5	0	5	13	6	8	8
3091	0	3	1	3	5	3	2	8	8
3098	8	5	4	7	8	10	14	8	3
3103	4	1	2	5	3	5	7	8	0
3115	0	3	3	9	2	10	11	6	10
3122	0	2	0	9	6	9	6	8	18
3124	1	0	2	5	9	8	6	4	4
3125	0	0	1	12	6	6	1	10	9
3126	0	1	1	9	2	3	6	6	11
3142	0	0	4	10	3	9	6	8	5
3148	0	0	4	7	7	6	1	7	7
3182	2	4	0	3	1	8	6	8	8
3192	0	1	0	5	1	7	6	5	13
3210	4	8	5	7	7	8	6	8	8
3224	2	2	1	7	7	8	4	9	9
3225	1	2	1	11	4	3	1	6	3
3230	0	3	2	4	4	5	2	5	7
3242	6	3	4	7	5	12	1	5	2

Table IIIb:- continued.

line/ cultivar number	Cycle number								
	5			6				7	
	Growth stage								
	29	32	39	27	35	29	50	28	31
4033	3	1	0	0	0	0	0	8	8
4034	0	0	1	7	7	8	6	8	8
4051	1	0	0	2	1	1	6	4	1
4054	0	0	0	7	7	8	6	8	8
4066	0	0	1	8	18	16	6	8	8
4072	1	0	0	0	0	2	0	4	1
4084	0	0	1	1	7	8	6	2	12
4085	0	3	0	0	0	1	1	2	2
4090	0	2	0	2	2	0	2	8	8
4091	9	0	0	0	4	1	6	8	8
4092	0	0	0	7	0	1	1	5	8
4093	0	0	0	16	14	9	6	8	8
4094	0	0	1	3	2	2	6	8	8
4101	2	3	1	0	6	8	2	8	8
4114	1	0	2	10	17	28	6	1	3
5006	7	6	4	10	9	10	18	14	7
5010	1	0	0	7	7	10	6	14	8
5023	11	6	10	25	26	8	6	14	8
5135	6	1	1	8	4	8	4	26	2
5153	2	2	2	7	20	4	6	8	8
6032	8	3	4	12	13	22	10	16	8
6038	10	10	2	18	22	20	6	26	27
6040	11	6	5	27	18	23	6	26	20
6041	3	4	3	10	12	14	17	19	19
6042	1	0	1	6	21	12	15	20	10
6044	7	2	5	15	16	19	6	23	26
6045	7	7	0	15	14	8	6	2	8
6046	3	2	5	11	4	1	4	16	13
6054	0	1	2	6	24	9	14	12	8
6062	2	7	1	10	13	16	3	8	3
mean	3	2	2	7	7	8	6	8	9
S.E.D.±	2.3	2.2	2.4	2.9	3.7	3.3	4.0	4.3	3.3
(D.F.)	(205)	(200)	(171)	(98)	(100)	(79)	(16)	(34)	(37)

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